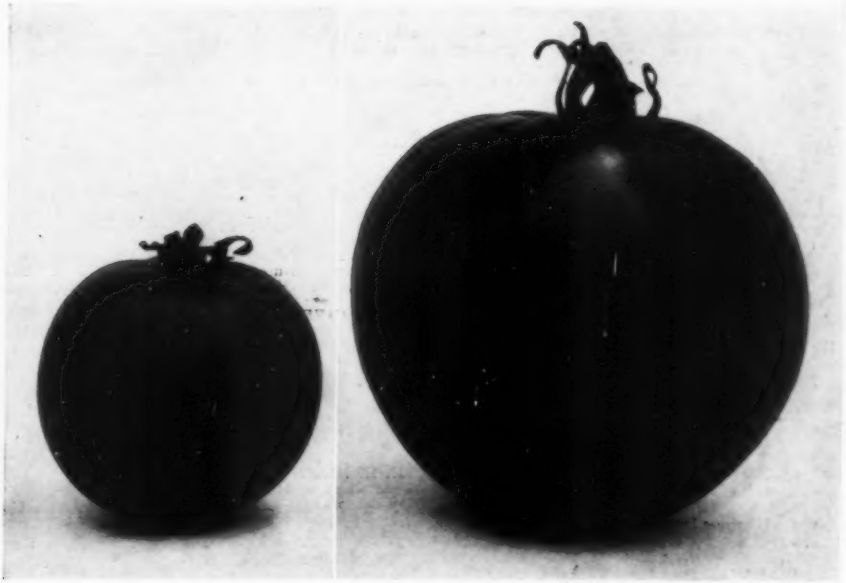
PEARL

The Journal of HEREDITY



"HALF-CHROMOSOME" AND NORMAL MARGLOBE TOMATOES

(SEE PAGE 433)

"In-Married" Nobility Have More Children Than "Out-Married"

Two Pedigrees of Inherited Predisposition to Hernia

A "Crippled-foreleg" Lethal Gene in the Horse

A "Trembling" Gene in the Field Mouse

Promising New Hazel-Filbert Hybrids

Where are the *Who's Who* Twins?

Love-Bird Genetics

VOLUME 27 NOVEMBER, 1936 NUMBER 11

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President Vice-President, W. E. CASTLE Secretary, C. E. LEIGHTY

Treasurer, J. H. KEMPTON

Legal Advisor, STANTON C. PEELLE

EDITORIAL BOARD

Managing Editor, ROBERT C. COOK

M. DEMEREC R. R. GRAVES L. C. DUNN

H. J. MULLER GEORGE M. DARROW PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.

LEON J. COLE, Professor of Genetics, University of Wisconsin.

G. N. COLLINS, Botanist, U. S. Department of Agriculture, Washington, D. C.

O. F. COOK, Bionomist in Charge of Breeding Investigations, U. S. Department of Agriculture.

DAVID FAIRCHILD, Agricultural Explorer, U. S. Department of Agriculture.

HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigations, U. S. Department of Agriculture.

SAMUEL J. HOLMES, Professor of Zoology, University of California, Berkeley, California.

C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.

N. D. C. LEWIS, Director, New York State Psychiatric Institute and Hospital, New York City.

J. H. KEMPTON, Bureau of Plant Industry, U. S. Department of Agriculture.

E. W. SHEETS, Chief of Animal Husbandry Division, Mississippi Agr. Expt. Sta., State College, Miss.

SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.

E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.

E. A. BESSEY, Professor of Botany, Michigan State College, East Lansing, Michigan.

A. F. BLAKESLEE, Plant Genetist, Carnegie Institution, Cold Spring Harbor, New York.

W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.

C. B. DAVENPORT, Carnegie Institution of Washington, Cold Spring Harbor, L. I., New York.

RUSSELL W. DUCK, Livestock Editor, *The Rural New Yorker*, Syracuse, N. Y.

E. M. EAST, Professor of Plant Genetics, Bussey Institute of Applied Biology, Boston, Mass.

R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.

C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.

C. M. GOETHE, Sacramento, California.

HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.

H. S. JENNINGS, Professor of Zoology, Johns Hopkins University, Baltimore, Maryland.

D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.

M. A JULL, Senior Poultry Husbandman, U. S. Department of Agriculture, Washington, D. C.

F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.

C. C. LITTLE, Director, Roscoe B Jackson Memorial Laboratory, Bar Harbor, Maine.

JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.

H. H. NEWMAN, Professor of Zoology, University of Chicago, Chicago, Ill.

T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.

JOHN H. PARKER, Professor of Agronomy, Kansas State College, Manhattan, Kansas.

JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.

A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.

GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.

EDMUND W. SINNOTT, Professor of Botany, Barnard College, Columbia University, New York City. (Representative of A. G. A. on Council of Amer. Assoc. for the Advancement of Science.)

W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.

CHARLES R. STOCKARD, Professor of Anatomy, Cornell University, New York City.

A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.

W. T. SWINGLE, Plant Physiologist in Charge of Breeding Investigations, U. S. Department of Agriculture, Washington, D. C.

W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.

H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.

H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.

E. N. WENTWORTH, Armour's Bureau of Agr'l Research and Economics, Chicago, Illinois.

P. W. WHITING, Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.

The

Journal of Heredity

Vol. XXVII NOVEMBER, 1936 No. 11

ORIGINAL ARTICLES

A New Lethal Factor in the Horse
By R. Prawochenski 411

Thoroughbreds Have More Children
By Frederick Adams Woods 415

New Filbert Hybrids
By C. A. Reed 427

An Inherited Tremor in Peromyscus
By R. R. Heustis and Elizabeth Barto 436

An American Pedigree for Woolly Hair
By Edgar Anderson 444

On the Genetics of Diabetes Insipidus
By S. G. Levit and L. N. Pessikova 445

Two Pedigrees Showing Inherited Predisposition to Hernia
By Luther S. West 449

Notes and Book Reviews

A Haploid Marglobe Tomato 433

Parenthood Institute Popular 438

The Budgerigar Cult (Rev. of Watmough, *The Cult of the Budgerigar;* Steiner, *Verebungstudien am Wellensittisch. Melopsittacus undulatus;* Crew and Lamy, *The Genetics of the Budgerigar*) 439

A Dynamic Theory of Personality (Rev. of Lewin, *A Dynamic Theory of Personality*) 441

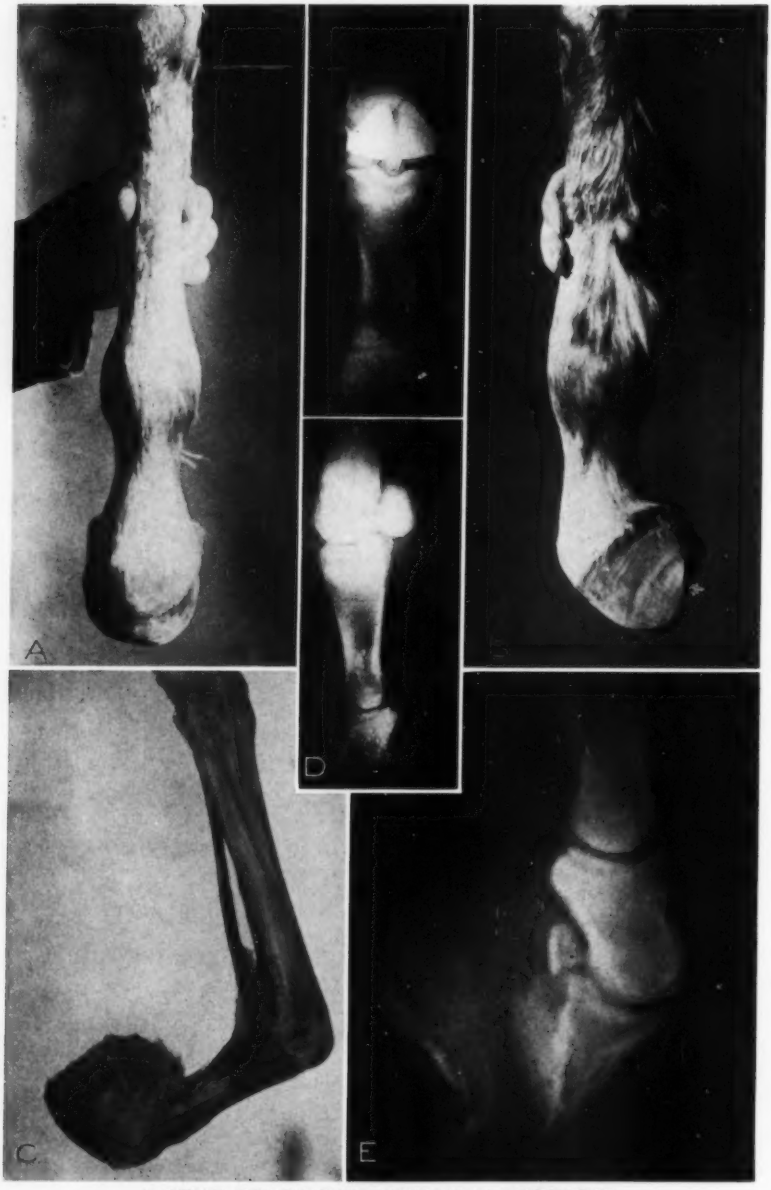
Some Questions Concerning Twins 442

Inheritance of Short, Stubby Hands 448

Books Received 455

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and Victor Building, Washington, D. C. Address all communications regarding editorial matters to Room 308, Victor Building, Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to the Victor Building, Washington, D. C. Entered as second class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the spepcial rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918. Contents copyrighted 1936 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office. $3.00 a Year. 35c the Copy. Prices of back numbers on application.

$3.50 a Year ***35c. the Copy***



A GENE PRODUCING CRIPPLED FORELEGS

Frontispiece

Nearly half of the foals of an Anglo-Arab stallion, Menzin, were born with deformed forelegs, as shown above. The character is indirectly a lethal because the crippled foals cannot stand, and would starve to death under natural conditions. They would be more quickly and humanely eliminated by the practical horse breeder. Above are shown front and side views of a crippled foreleg (*A, B*), a detail of the affected joints (*C*), and three X-ray views of crippled legs (*D, E*). The development of the rest of the body is normal and the hind legs are unaffected.

A NEW LETHAL FACTOR IN THE HORSE

R. PRAWOCHENSKI*

Zootechnical Laboratory of Jaguellonian University, Cracow, Poland

THE strenuous physical exertion necessary in all of the various ways that horses are used undoubtedly has had a definite influence upon selection of brood mares and stallions. This probably explains the small amount of data concerning lethal or sublethal factors found in the genotypes of horses described by various writers. This situation is in contrast to that in cattle, where a number of lethal genes have been found widely disseminated in some of the dairy breeds.

Yamane,[5] in Japan, described the first case of a lethal gene in the horse, associated with colic among the foals sired by the Percheron stallion, Superbe, which was imported from America.

Wriedt,[4] in his *Biologische Essays über Pferdezucht,* gives nothing except an example of so-called diminished vitality, with accompanying sterility, in the Frederiksborg Stud, in Denmark.

F. A. E. Crew and A. D. Buchanan-Smith collected in their valuable work, *The Genetics of the Horse,*[1] numerous hereditary pathological characters, such as bleeding, osteoporosis, roaring, spavin, polyarthritis etc., all of which are the result of so-called semilethal or sublethal genetic factors rather than of factors accurately classified as lethals.

No other case of a true lethal genetic factor in the horse has been reported up to the present time. It is true that from time to time we hear about some monstrous newborn foal, but they have no association with any particular strain of equine blood.

We are justified in classifying the crippling defect reported below as a true lethal factor because the foals are unable to stand, and would inevitably starve under natural conditions. Their elimination is just as sure, though more mercifully rapid, under the practices of the horse breeding farm.

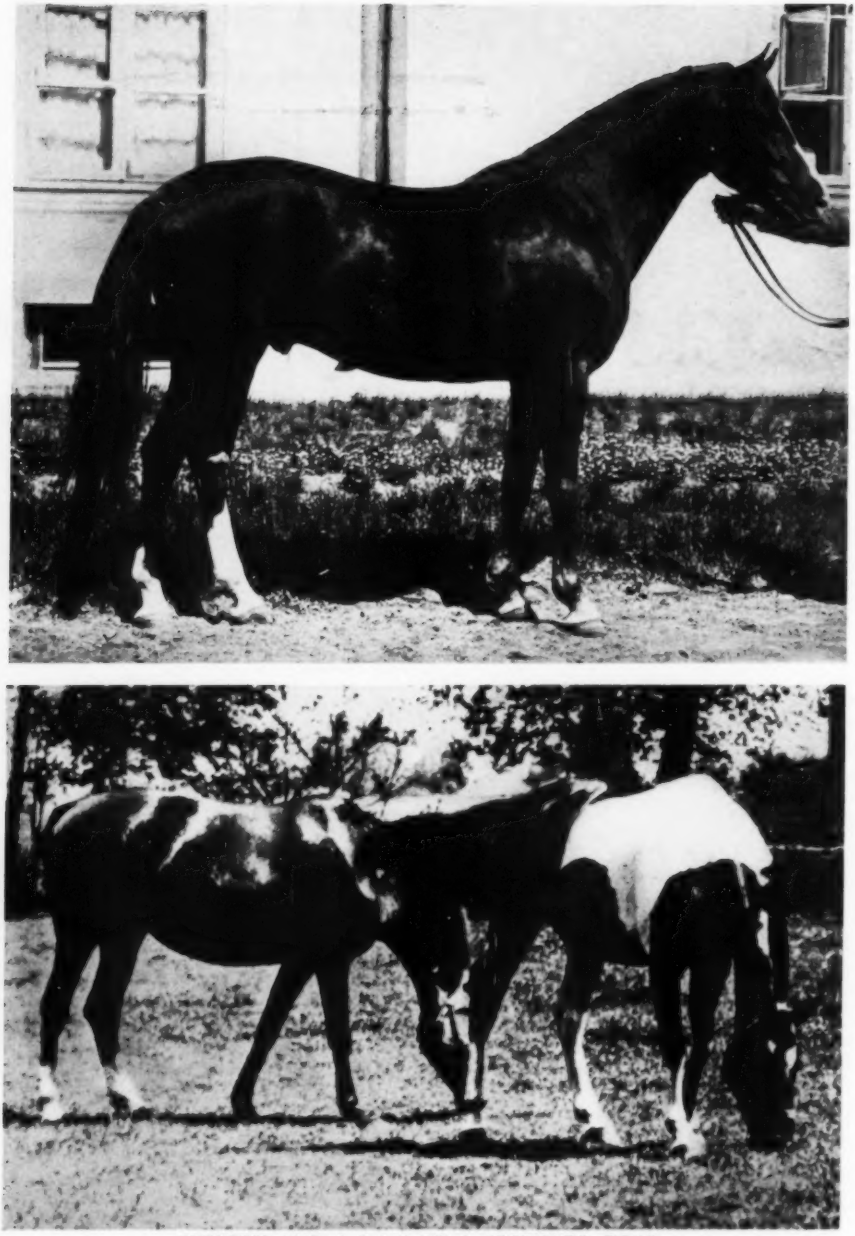
Therefore my interest has been aroused by a very striking phenomenon observed in a stud of half-bred horses, mostly of the cross Thoroughbred × Arab, on the estate of Skrzydlow, at Aurelow, near Czestochowa, Poland.

This stud farm received from the Government Stallions Depôt, for the season of 1933, an Anglo-Arabian horse, Menzin-N554, inscribed in the Polish Studbook of Half-Bred Horses, V. I. P. 204 (see Figure 1). Born in 1928, as can be seen from his pedigree Menzin is of three-fourths Arab blood. He is a chestnut, 15 hands high at the withers, good looking, and his conformation is without defect except, perhaps, some signs of too lymphatic a constitution for a light horse.

The next year, 1934, nine foals sired by Menzin were born at Skrzydlow stud. Among them six individuals were quite normal, but three showed lameness unusual in newborn foals. One or both forelegs of these three colts were defective, with typical crooked, immobilized phalangia. The foals that could not stand at all were destroyed. The foal with only one crippled foreleg was helped by a stud-groom for several days to stand and to suck the mother, but the crooked leg showed no improvement. The cases, although of a very striking nature, were regarded as being due to coincidence, and many suppositions concerning environmental influences of feed or surroundings were made.

The general standing of the stud-farm is high, and every year it sells its

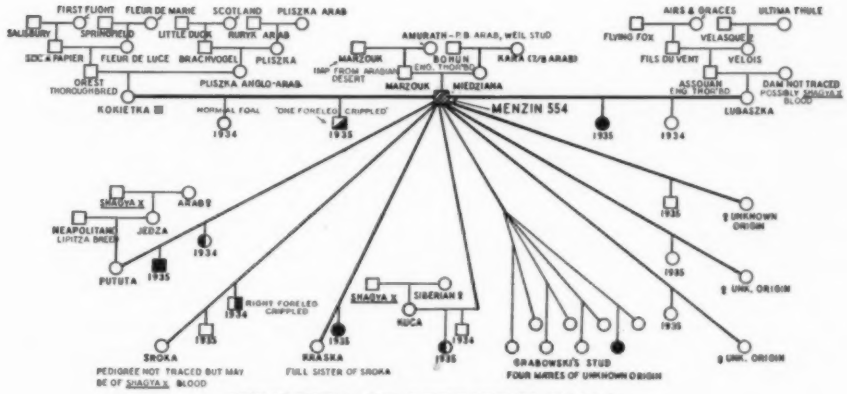
*My thanks are due to Dr. med. vet. Guzek and to my assistant, Dr. Slizynski, for valuable help in examining the anatomical character of the lameness and for making the photographs and X-rays. Also I express my thanks to the owner of Skrzydlow stud-farm, Mr. A. Danilczuk, for all particulars concerning the foals, and to ing. agr. W. Pruski from the Ministry of Agriculture in Warsaw, who kindly gave all information about the pedigrees of Menzin and of the brood mares mated with this unfortunate stallion.



MENZIN AND A DAM OF A CRIPPLED FOAL

Figure 1

Above is shown Menzin, a chestnut Anglo-Arab stallion born in 1928, and used at the Skrzydlow stud in 1933 and 1934. He was without visible defect, and his ancestry as far as known was normal with respect to conformation of the foreleg. Below are shown two mares of this stud on pasture in 1934. The piebald mare (Sroka) bore a crippled foal sired by Menzin. (See Figure 2.) Photographs by courtesy of A. Danilczuk.



PEDIGREES OF DEFECTIVE FOALS

Figure 2

All the crippled colts born at the Skrzydlow stud were sired by Menzin during 1933-34. Their pedigrees are given in summary here. There is no close relationship known between the mares, though several of them may trace to the Arab stallion Shagya X. If this defect is conditioned by a single recessive gene it must be widespread and should have appeared elsewhere. The fact that Menzin is himself normal and had normal ancestors seems to rule out a dominant character. It is clear therefore that this pedigree does not make possible a complete analysis of the mode of inheritance of the defect.

three-year-old colts, at highest prices, to the cavalry remount. The foals are reared under excellent conditions, both they and the brood mares having plenty of exercise, good pastures, and a well balanced diet.

In 1935, 13 foals sired by Menzin were born. Again, nine of them were normally developed and four were cripples. On a neighboring stud-farm, owned by Mr. Grabowski, where Menzin also covered some mares in 1934, four foals were born—three normal and one cripple. All cripples had the same crooked phalangia, giving the same aspect of immobilized termination of the foreleg, with an undeveloped shortened hoof which resembled a small apple (see Frontispiece).

It must be emphasized that never before had the brood mares at Skrzydlow brought forth any such crippled foals. As can be seen from the list of progeny under the name of each brood mare (see Figure 2) it is exclusively the foals sired by Menzin that are cripples. No other stallion used before at Skrzydlow and mated with the same brood mares sired foals with such a defect.

It seems a logical conclusion that Menzin must have in his genotype a pathological factor related to the lameness under discussion. The only question would seem to be one of the mode of inheritance involved.

In connection with this character of lameness, which has been examined from an anatomical point of view, no signs of rickets or arthritis, or any other bone disease have been found (see Frontispiece). There seems to be a lack of balance between the tendons (flexors and antiflexors) or else unusually short flexors. Associated atrophy of corresponding muscles in higher parts of the forelegs also is not excluded. Of course, from a genetical point of view, it is of no importance whether the localization of the cause of crippleness is in the central nervous system or elsewhere in the body.

The hind legs, including the feet and hoofs, were quite normal, as were the general conformation and development of the foals.

Perhaps we have here an analogy of the case described by Mohr[2] in 1926, when he found in a Norwegian human family a lethal factor in relation with

"brachyphalangia" in new-born children.

Coming back to the problem of the mode of inheritance, we must first compare the pedigrees of Menzin and his mates. As can easily be seen, no close relationship of their blood can be found (see Figure 2). It is true that some strains of Arab blood undoubtedly have some common ancestors, but they are very remote in the pedigrees of Menzin and these brood mares.

At any rate, I call attention to the fact that it is the blood of an Arab stallion, Shagya X, born in 1899 at Radowce stud (once the property of the Austro-Hungarian government), which we meet most frequently in the pedigrees of the brood mares, who brought forth the cripples. It is not improbable that some of these brood mares whose origin was not traced also have the blood of Shagya X in their veins, because Arabian blood was very popular in the neighborhood of Skrzydlow.

During the Austrian occupation and in the post war-time stallions from Radowce stud were used at the temporary breeding stations by peasants and farmers, but in many cases we do not have exact data concerning their progeny.

Generally the brood mares at the Skrzydlow stud are of a type distinctive of half-bred horses (see Figure 1).

As to the English Thoroughbreds in the pedigrees of Menzin and his mates, no common ancestor can be found, at least during five or six generations.

Therefore it is obvious that we are not dealing with a simple recessive gene, especially because of the very frequent occurrence of the defect, the ratio of cripples to normals during the two breeding seasons being 8:18. Of course Menzin has now been removed from the stud, and we will be unable to procure further data concerning this character.

At any rate, I requested the owner to feed the crippled filly artificially and to endeavor to rear her in order to mate her with Menzin or with a crippled horse reared in the same way. But, as is easy to understand, such an undertaking is very difficult of realization under the conditions of a private farm.

Discussing with Professor Moczarski* the phenomenon of such a high percentage of inheritance of the lethal, or at least sublethal, gene, I was told that perhaps we have here a case of mutation. If this is so, then my interlocutor must suppose a case of mutation in the sense of gene deficiency—when a recessive gene, having lost its allelomorphic partner in the homologous chromosome as a result of mutation, behaves as a dominant. Such a phenomenon was found in Drosophila.[3]

Of course it can be discovered and other explanation associated with the supposition of numerous genetic factors involved here and bringing a pathological character of inheritance as a result of special gene arrangement. It is not improbable that several distinct genetic factors with a pathological tendency exist in the genetype of the horse. These are manifested as a more or less expressed predisposition to lameness, depending solely upon the number of genes involved in a given inheritance. The above described phenomenon of a complete crippling among new born foals must be connected with a special coincidence of all genetic factors of this kind, from both the paternal and maternal side.

At any rate the case here presented of such an unusual lameness connected with the necessity of destroying newborn foals is worthy of the attention of breeders and geneticists.

Literature Cited

1. Crew, F. A. E., and Buchanan-Smith, A. D. The Genetic of the Horse. *Bibl. Genetica*, VI, p. 141. 1930.

2. Mohr, O. Ueber Lethalfactoren. *Zeitschr. f. Induct. Abst. und Vererb.* 1926.

3. Sinnot and Dunn. Principles of Genetics. London. 1934.

4. Wriedt, C. Biologische Essays ueber Pferdezucht und Pferderassen. Berlin. Paul Parey, p. 37. 1929.

5. Yamane, J. Ueber die Atresia Coli, eine erbliche Darmmissbildung beim Pferde. *Zeitschr. f. Ind. Abst. und Vererb.*, 46, 188. 1927.

*My colleague from the University of Poznan.

THOROUGHBREDS HAVE MORE CHILDREN

Aristocracies Form and Persist Through Marriage Selection

FREDERICK ADAMS WOODS

ALFRED RUSSELL WALLACE stated in his *Studies Scientific and Social:* "In one of my latest conversations with Darwin he expressed himself very gloomily on the future of humanity, on the ground that in our modern civilization natural selection had no play and the fittest did not survive." Wallace himself insisted there are forces to be counted on for the amelioration of the race, one of which is the process of *elimination* "by which vice, violence and recklessness so often bring about the early destruction of those addicted to them." He does not, however, foresee mental evolution.

It is gratifying to know that now, after so many years, we have many proofs of mental evolution at least within the upper classes. Among royal families mental qualities have been proved to be correlated with moral.[16,17] The brothers and uncles and cousins who rank the highest in intellect, virtues, force and ambition, have the largest number of adult offspring.[17] The same is true for college graduates, as was first proved in 1926 by the author.[23,24] The upper two per cent of Nordic population does not have as many children as the lower ranks. This, as far as it goes, is contrary to evolution. But, if *within* the two per cent, a constant spouting upward can be seen, and if this small superior class is being separated biologically from the remainder, then there is a present-day advancement of inherent brain power. Nowhere in organic evolution do we expect to find that *all* species or *all* subvarieties advance.

Psychological and social selection, assortative mating and differential fertility working for betterment have been found for royal families, British peers, Harvard and Yale graduates, and men of science. In the present article I will show a new reason for believing in the natural conservation of aristocracy. It is part of the same general theory as to how aristocracies form, move toward exclusiveness and persist.

How Aristocracies Form

Some fifteen years ago, at the Second International Congress of Eugenics held at the American Museum of Natural History, New York, I presented evidence proving an actual formation of aristocracy in the United States. It was shown that old New England families had, in spite of democratic institutions, become gradually separated into two special classes more and more removed from each other in terms of wealth, intermarriage and worldly eminence.

The royal families of Europe and old noble families of Great Britain show the same tendencies. They move towards isolation and self-perpetuation. They do not become less aristocratic as time goes on and they do not tend to degenerate or to disappear. In fact, there is much evidence that the whole world is, and always has been, at least attempting to divide biologically into castes—especially into two divisions, an immense proletariat, and a few sexually selected individuals who tend to intermarry within their own circle and slowly rise, drawing themselves up and thus dominating the inferior.

This view is contrary to popular belief, but it is nevertheless a fact. Research shows that leaders come less and less, as time goes on, from the families of the lower classes. Of course there are exceptions, and today we have examples of one or two dictators, the

product of sport variation. Napoleon was such, and there have always been born occasionally geniuses like Lincoln and Franklin from undistinguished stock. The majority of eminent men, however, come and always have come from families well above the average, as is proved by the historiometric studies of many investigators.* But the most remarkable point in this connection is that in Great Britain where democratic institutions, popular education, and political religious freedom have all markedly advanced, the ratio of notable men from the lower classes has actually declined.

The *Dictionary of National Biography* shows that prior to the Nineteenth Century 11.7 per cent of its most eminent men were sons of skilled or unskilled laborers. This declined to 7.2 per cent during the early part of the Nineteenth Century, and for a more recent period (second supplement) it has declined to 4.2. It is a manifestation of the same sort of social tendency shown in the genealogical and economic history of New England families.†

This tendency of social conification or social aristogenesis is not always at work. It was not at work in Russia at the close of the Great War. It was not at work in France in the Reign of Terror. There are such things as revolutions. Also patrician breeds may disappear for perhaps many reasons—we do not as yet know why. They went from Ancient Greece and from the Rome of the Empire, and from the intellectual Italy of the Renaissance, but their reappearance in other locations and their inevitable engenderment is a subject treated in several of my various contributions. Also I have new evidence to prove that there are internal forces of a biological nature which determine the persistence of superior breeds.

It seems that there are two chief factors which constantly hold old and aristocratic families to form. As these forces (or tendencies) have never been noticed—as far as I know—and as the data and conclusions are novel, I will present this evidence now.

Everyone knows that royal and noble families have a habit of marrying within their own sets and are inclined to have a great many children. Such are obvious causes of their self perpetuation, but the point that I have recently discovered is that when they marry below their own social position *they do not have large families at all.* Indeed almost without exception the number of offspring is strikingly small. There can be no doubt as to the existence of this real difference. The averages for all small groups of ten or a dozen marriages exhibit the greatest contrasts. Royal and noble marriages *inter se* produce about three adult offspring on the average. The same with non-noble women never average much more than one.

The following figures will be presented in as brief a way as possible con-

*Some researches which, in one way or another, prove that the upper social classes have very much more than their share of natural mental capacity are those of Galton,[4,5,6] de Candolle,[1] Odin,[6] Ellis,[2] Woods,[17,19,20] etc., and the Whethams' study of the British peers; Yerkes, Brigham and others in their analysis of the United States Army mental tests; Roswell H. Johnson's study of business men; McDougall on the correlation of intelligence and social status, and Woods' *Conification of Social Groups*,[23] both the latter published in *Proceedings of the Second International Congress of Eugenics.*

Ireland,[7] Stoddard,[18] and Wiggam[14,15] contain discussions of and references to the theory of social conification. More recently Terman and Cox[3] have brought added proof that social position is highly correlated with inborn intelligence and also fresh evidence for the scientific accuracy of historiometry.

†A more extended discussion is given by Ireland[7] and Wiggam.[15] The first two percentages, 11.7 and 7.2, were found by H. Ellis.[2] The third, 4.2, was found by myself in 1919. It convinced me that the two differences noticed by Ellis were not mere chance but part of a far-reaching biological and sociological tendency. In order to plot a "curve" at least three points are necessary. A. E. Wiggam is therefore in error in stating "It was Havelock Ellis who first noted the existence of this progressive decline in the production of eminent men by the laboring classes, but it was Woods who suggested its cause and significance."

sistent with the need of giving raw data in a form fit for verification. The numbers are drawn from recent editions of the *Almanach de Gotha*, 1923, 1933, and 1935. This Almanach is divided into two divisions. The second part is *diplomatique et statistique* and does not concern us here. The first part is called *Annuaire Genealogique*. It deals, as it says, with the present sovereign houses and former sovereign houses, with the German *médiatisées* families and the majority of the non-sovereign ducal and princely houses of Europe. This entire section (600 pages) is the basis of this present article, and another which I hope will follow soon, showing the antiquity and fecundity of the actual sovereign houses.

I will give first the results from the *Deuxième Partie Genealogie des Maisons seigneuriales médiatisées d'Allemagne*. They are the families of the princes and counts who formerly belonged to the Holy Roman Empire. They are considered as having the "rights of equality of birth with the sovereign houses." The names are given in the footnote.* Following the names are the dates (Arenberg A. D. 1047, for instance) when the family first emerges from the mists of historic uncertainty carrying a title verifiable in male-line descent to the present day. The next figures, 7-1, mean that for recent princes and dukes of Arenberg there have been seven royal, or noble, marriages and one non-noble. The next figure, 20, means that the entire family, males and females—not including wives —totaled twenty in the 1923 edition.

It can be seen at a hasty glance that most of the families are very ancient and few of them are small—only one, namely Stadion, being under five. Several are large—Castell 54, Hohenlohe 121, Isembourg 54, Lobkowicz 66, Platen 56, Schönborn 48, Schönbourg 61, Solms 119, Stolberg 140, Turn-et-Taxis 44, and Windisch-Graetz 51. Their ages can be best appreciated by viewing them as a frequency group. (See Table I.)

The way they group themselves around the end of the twelfth century is truly remarkable. It indicates to some extent the homogeneousness of these Germanic princely families. Their origins and developments have been very similar.

If old and aristocratic families died out because of their age we should not expect such a showing. They started hundreds of years ago. There were then just twice 54 or 108 individuals. There are now by actual count 1,677. In this JOURNAL I have already shown[27] the perpetuation of old English and Scotch families. More than half the British peers (334 out of 622 in 1921) are continually aristocratic in the direct male lines as early as A. D. 1450. It is the same on the Continent, only more so. Only three of these fifty-four families,—Kuefstein 1602, Esterhazy 1613, and Rosenberg 1621, date their earliest noble ancestor as late as the first half of the seventeenth century. Fugger, the famous princely house of commercial origin, dates from 1507. All

*Arenberg (Ligne) A. D. 1047, 7-1, 20; Auersperg 1050, 11-1, 34; Bentheim 1191, 5-2, 18; Bentinck 1233, 4-2, 22; Castell 1057, 14-0, 54; Colloredo-Mannsfield 1302, 4-2, 19; Croy 1178, 21-22, 75; Erbach 1148, 11-3, 21; Esterhazy 613, 2-0, 7; Fugger 1507, 8-2, 30; Fürstenberg 1070, 6-1, 17; Giech 1125, 1-0, 5; Harrach 1338, 12-0, 36; Hohenlohe 1170, 33-6, 121; Isembourg 1042, 13-2, 54; Khevenhüller 1080, 6-0, 14; Könegsegg 1171, 2-0, 10; Kuefstein 1602, 2-1, 8; Leiningen 1080, 6-4, 14; Leyen 1272, 1-0, 9; Lobkowicz 1459, 12-1, 66; Looz 1300, 3-3, 7; Löwenstein 1476, 8-1, 36; Metternich 1320, 2-0, 6; Neipperg 1241, 3-0, 14; Oettingen 987, 6-0, 20; Ortenbourg 904, 3-0, 10; Pappenheim 1131, 8-0, 21; Platen 1255, 15-2, 56; Pückler 1478, 4-3, 18; Quadt 1313, 5-1, 20; Rechberg 1179, 5-0, 27; Rechteren 1230, 8-1, 19; Rosenberg 1621, 5-1, 16; Salm-Supérieur 1173, 7-0, 22; Salm-Inférieur 1147, 10-0, 33; Sayn 1128, 14-5, 39; Schaesberg 1335, 4-0, 14; Schlitz 1100, 2-0, 12; Schönborn 1284, 10-1, 48; Schönbourg 1130, 17-1, 61; Schwarzenberg 1172, 4-0, 27; Solms 1129, 30-3, 119; Stadion 1197, 0-2, 3; Starhemberg 1150, 2-0, 8; Stolberg 1210, 35-1, 140; Thurn-et-Taxis 1117, 16-2, 44; Torring 1100, 1-0, 5; Trauttmansdorff 1308, 10-1, 38; Waldbott 1138, 1-5, 15; Waldbough 1170, 10-1, 33; Wied 1226, 2-0, 11; Windisch-Graetz 1242, 26-1, 51; Wurmbrand 1130, 25-3, 30.

the others are extremely ancient, and it is curious that these very ancient houses now average many more representatives at the present day than the four more recent ones who are comparatively unimportant biologically, having in each case a small number of living representatives. Statistics concerning their offspring will follow later.

Italian and French Families

I have also made a careful study of the Italian princely families as they appear in the *Almanach de Gotha,* 1923 and 1935. It is the same story. When the marriages are with nobles the number of offspring is nearly three. When with non-nobles, whether they be Italian, American, English or otherwise, the number on the average is one and a half. Table II shows how strikingly this is true. There are 56 of these princely houses,—a list too long for detailed statistics to be given here. The table has therefore been broken into ten groups according to their alphabetical arrangement. In Table II every subgroup shows the marked difference, always in favor of the thoroughbred unions. Only once in the case of the initials O-P do we see the figure for the cross-breds rising above two, and that is only for a small group. The cross-bred progeny are not sufficient to maintain the stock. The average is 1.49, just about half of 2.92, the average number of offspring of noble with noble.

The differences are even more striking among the Germanic mediatized princely families, and this is as it should be since their social rank is somewhat higher. Their rights of birth are considered as equivalent to reigning sovereign royalty. Therefore if anyone marries a commoner his mesalliance is more definitely a stepping out from the fold. Often times they lose cast en-

TABLE I. A View of the Distribution of the Age-Origins of Germanic Royalty, as a Frequency Group. If their age-origins are arranged in a table of frequencies it runs as follows:

Centuries	IX	X	XI	XII	XIII	XIV	XV	XVI	XVII	XVIII	XIX	XX
Total 54	—	2	7	22	9	7	3	1	3	—	—	—

TABLE II. Showing How Few Are the Offspring of Italian Noble Mixed Marriages in Contrast with the Thoroughbred.

	Thoroughbred			Mixed Marriages		
	Number of Marriages Noble with Noble	Number of Offspring Noble with Noble	Average Number of Offspring	Number of Marriages Noble with Non-Noble	Number of Offspring Noble with Non-Noble	Average Number of Offspring
A	6	23	2.4	1	—	0
B	34	86	2.5	12	15	1.2
C	41	110	2.7	22	35	1.6
D-F	10	19	1.9	8	10	1.2
G-L	18	38	2.1	8	14	1.7
M	13	60	4.6	—	—	
O-P	16	54	3.4	4	10	2.5
R	20	73	3.6	12	13	1.1
S	10	21	2.1	3	5	1.7
T-V	7	27	3.9	4	8	2.0
Totals	175	511	2.92	74	110	1.49

Italian princely families with dates of their earliest nobility: Altieri 1396, Angera 1445, Antici-Mattei 1249, Avellino 1560, Barbiano 1365, Biscari 1398, Boiano 1536, Boncompagni-Ludovisi 1133, Borghese 1238, Brancaccio 1645, Caetani 875, Camporeals 1304, Caramancio, Canneto 1625, Carini 1403, Cassano, Castagneto 1300, Centurione 1360, Cerami 1132, Chigidella Rovere-Albani 1200, Cito-Filomarino 1753, Colonna 1100, Corsini 1371, Doria-Pamphili 1335, del Drago 1133, Forano 1059, Frasso 1200, Ginori XV Cent., Giustiniani-Bandini XVI Cent., Gonzaga 1150, Hercolani 1429, Linguaglossa XIII Cent., Massimo 950, Molfetta 1340, Montignano 1508, Musocco 1277, Odescalchi 1676, Orsini 1190, Paterno 1295, Pignatelli 1102, Rospigliosi 1667, Ruffo 1200, Ruspoli 1500, Sacchetti-Basbarini 1633, Sant'Angelo dei Lombardi 1518, Scilla 1777, Serra 1574, Soragna XVI Cent., Spinosa XII Cent., Strongli XIII Cent. ?, Trabia XIII Cent., Valsavoia ?, Vicovaro ?, Villafranca 1300.

tirely, or have to content themselves with a lower title. This is the second way that the type is maintained. It will be discussed later.

From 1875 to 1899, 133 noble marriages produced 396 mature offspring, or an average of 2.98. At the same time there were thirteen non-noble marriages. These resulted in 19 mature offspring, or an average of 1.46. From the years 1900 to 1913 there were 111 noble marriages. There were 338 offspring, or an average of 3.05. At this time there were twenty marriages of nobles with outsiders with only fourteen offspring, or 0.7 per marriage, for the average.

During the war years 1914 to 1918, thirty-two "pure-bred" marriages brought 107 offspring, an average of 3.34. During this period five marriages with commoners brought only four children, or 0.8 for each. Again from 1919 to 1930 there is much the same record. The 121 noble marriages produced 268 offspring, an average of 2.21. The non-noble wedlocks are thirty-seven in number and result in thirty-five offspring or a little less than one. Naturally, latest marriages furnish families as yet uncompleted, but the ratios remain always about the same,—namely two to one.

Just after the Great War there was apparently a rush to marry. During the years 1920, 1921, and 1922 there was an average of twenty-one marriages with nobles as wives, whereas during the war it was only 6.4. There were thirteen marriages with non-nobles during these same years and this is considerably above the number expected.

Does it mean a social change? It is particularly interesting to try to determine how much effect the sweeping decrees, which directly after the war abolished all titles, really had on the exclusiveness of these people. At first glance it would seem to be noticeable.

From the year 1900 to 1913 there were 111 marriages of nobles with nobles against twenty with outsiders. In other words, 15 per cent took non-noble wives. This same ratio for the war years is five to thirty-two or 13.5 per cent. This rises to 22.3 per cent for the years after the war,—39 out of 177 were of the mixed variety.

Still I do not believe there is sufficient evidence to show that the revolutions in Germany, Austria, and in the Republic of Tchecoslavakia made any change. In Austria, and in the Republic of Tchecoslovakia titles of nobility were prohibited by the laws of April 3, 1919, and December 10, 1918. In Prussia the titles of Highness (Royal, Serene, or Most Illustrious) were abolished by the law of July 23, 1920. This is a considerable environmental change and well might be expected to show a result. But if it has made the group less exclusive in marriage selection it has not made the habit grow. There are proportionately less mixed marriages during the second half of this post-war period than in the first. Also during the latest tabulated years 1930-32 there were only three outside marriages in twenty-six,—a return to the pre-war standard. The figures are too small to give positive disproof but at least they say "Not proved and improbable" against the radicals who may have thought that by reducing the old aristocracy in wealth and tinsel glamour they could force them down biologically or make their families any newer. It was the same in France after the abolition of the *Ancien Regeme*. Many of the "aristocrats" were killed or driven out of the country by the Revolution of 1789 and forced to emigrate to Switzerland, Italy, England, the West Indies or the United States. But those who remained, continued, and still continue to a great extent, to hold themselves socially aloof from the bourgeoisie. A scanning of the pages of the *Almanach de Gotha* shows that the marriages (especially of the daughters) are usually with their own class.

As this is an important point at this stage of my argument, I have made a careful count in alphabetical order of all families, dating prior to the Napoleonic era, of the French nobility of

the higher ranks. Only the families of the important dukes and princes are listed in this book,—much the same as for the Italians. The names of the families and dates of their earliest claims to nobility are given in the footnote.* The antiquity of most of them is very evident. Here are considered only those taking origin prior to the French Revolution. There are also in the *Almanach de Gotha* thirteen modern families, but these are not included in my statistics.

The sub-groups show the following ratios of non-noble marriages for the sons. A-B, 6 in 39; C-H, 6 in 31; L-M, 12 in 65; N-S, 19 in 70; T-W, 7 in 15; total, 50 in 220. This is 22.5 per cent and is actually lower than that for the Italian non-noble marriages which is 29.7 per cent—and this in spite of the fact that Italian nobility is officially recognized by the sovereign, the court and the government. Also in France the thoroughbred unions produce many more offspring than the others. Forty non-noble marriages resulted in only forty-two offspring. One hundred and forty within their own ranks produced three hundred and seventy-one,—an average of 2.65 against 1.05—a great difference.

Daughters Very Exclusive

As stated above the daughters of these families tend to marry outside the titled nobility even less than the sons. The same is true among all the royal and noble groups here studied. Among the French this is about 3.8 per cent (6 in 149). For the Italians I have no precise figures. The number is certainly very small. An exact count for the daughters of the princely houses, mediatized (non-sovereign royalty), mostly German and Austrian, shows from 1878 to 1932 only 29 in 352, or 8.2 per cent against about 20 per cent for their brothers. It is impressive that the daughters of the old noble pedigreed clans in spite of outward forms of democracy, commercial and political upheavals, still adhere—or their parents still adhere, or all still adhere—to the old feelings. It seems instinctive more than worldly. When they do marry other than nobles they generally marry professional men. In the case of the nineteen mediatized princesses and countesses who since 1918 have married non-nobles, ten were with doctors or lawyers, or young military officers, or government officials. Two married chemists and one married an artist. Thus there is evidence that they do not often marry men engaged in trade or finance. Apparently they do not marry for money. Maybe it is assortative mating. Maybe it is love. One of the strong motives for love noted by Stendhal in his well known writings on this subject was social position,—a correlary of pride. Its importance in history, in romance and in human evolution has, in my opinion, been very much overlooked.

Demotion in Rank

The survival of the type is—we have seen—maintained by the decreased net fecundity of those who marry outside the royal fold. This permanency is also secured by the simple process of casting outward or at least downward, many (though not all) of those who make misalliances. Such changes in rank are not always technically or legally insisted upon, but there are, in

*Old French families, ducal and princely, listed in the *Almanach de Gotha* for 1923. They are arranged in the order of their antiquity. The dates indicate the first authentic records of their nobility mentioned in the Almanach. Several of the families are undoubtedly older than the dates here recorded: Choiseul-Praslin 937, Lucinge 981, La Rochefoucauld 1019, Fezensac (Montesquiou) 1030, Polignac 1031, Rohan-Chabot 1040, Talleyrand et Sagen 1066, Harcourt 1074, Clermont-Tonnerre 1080, Bauffremont 1090, La Trémoille 1096, Sabran-Pontheves 1108, Luynes 1150, Rarécourt 1170, San Fernando-Luis (maison de Lévis-Mirepoix) 1203, San Lorenzo (maison de Dampierre) 1203, Broglie 1254, Beauvau-Chaon 1265, des Cass (maison de Pérusse) 1281, Lorge 1305, Umbriano 1323, Cramont (maison d'Aure) 1381, Uzès (maison de Crussol) 1483, Chalais before 1508, Brissac (maison de Cossé) 1560, Richelieu (maison de Jumilhac) 1596, Caylus 1756, Wagram 1763.

the second division of the *Almanach* a considerable number of such demotions. For example, there are five cases in the princely family of Hohenlohe,—not so very many perhaps considering that the total family population is 121. Two of these were reduced to the rank of counts, and two to that of barons. One is now included in the handbook of Adeligen Häuser (noble families) a less important list. The families of Erbach, Castell, and Sayn Wittgenstein have lost seven members by these unconventional unions; Bentheim, Fugger, Furstenburg, Isembourg, Looz, Löwenstein, Pückler, Quadt, and Pappenheim each one. It is needless to point out that the biological effect is a process of weeding out, similar to natural and artificial selection. It tends to maintain a conservative and isolated breed. Those who conform to the laws and customs of the family are accepted. The rebels either voluntarily leave or are driven out or down.

In quoting my researches on social conification, in connection with this tendency of biological isolation, Popenoe and Johnson[10] make the following observations with which I heartily agree:

> If one takes a long view over the past, [geographical] isolation seems to be more important than any other single factor in evolutionary differentiation, because it tends to concentrate the lines of descent and thereby to produce differences of gene-balance. While it has been a conspicuous factor in human evolution, even in contemporary times, it will probably be less and less active as freer movements of population take place. On the other hand, geographical barriers are by no means the only ones, or the most important ones, that create isolation in this sense. Racial, linguistic, religious, social or economic barriers have been highly effective and it is by no means impossible that future evolution will see some of these barriers persisting longer than is generally expected. Many naive views are expressed about the likelihood that all mankind will eventually become homogeneous. This is not the ordinary course of events in any species with a wide range, and there is no evidence that it is occurring in man. In fact, the opposite tendency seems to be found in many directions, as manifested for instance in more definite stratification of society along social and economic lines, in a long-settled community.[12] It is not at all impossible that social and psychological barriers will function more effectively in the future than they do now, as equalitasian ideas are gradually dropped.

War and Royalty

Much has been written *pro* and *con* on war and eugenics. Most of it impresses one as being put forth by people who already are sure that war is dysgenic and they are going to come in on the glory of so proving it. Like discussions concerning racial questions, the emotional element is here so strong that search for the truth is sorely hampered. It is usually asserted that war kills "the best," that it kills more generals and more staff officers and more field officers than it does average men or inferior men. If war kills especially "the best," then there is some sort of exception in the case of royalty. Either royalty are not of "the best" or modern methods of warfare have altered the ratios of former generations; or perhaps the assertion that war tends to kill the best has never been correct.

There were six princes killed in the Great War out of a total population of about 2,500 of the second group of royalty. These were of the families of Wurmbrand, Trautmansdorff, Waldburg, Arenburg, Salm, and Thun. This would give a percentage of .24 of one per cent. These mediatized princes are nearly all German or Austrian. If we compare this with the German officers of all sorts and German men in the ranks it turns out that the percentage kill is less than one-tenth as great as that for non-royal Germans! According to K. Saller[11] of 410,000 officers of all sorts in the German army, 13.8 per cent died; and of 13,000,000 men in the ranks (including non-coms) an almost equal percentage, 13.0 per cent died.

If the population of Germany be taken as sixty million it would mean that about twenty-two per cent of the population were engaged in the World War and about 2.9 per cent of the population were killed. The losses for officers and for men is now about the same for each. In the Franco-Prussian War the losses for officers were distinctly

greater than for enlisted men, but as Popenoe and Johnson state,[10] (from whom my data are taken) this change is probably due to the new military technique. A similar situation is found by H. R. Hunt in regard to casualties in the American army for the recent war. But even in the Franco-Prussian War the loss for generals, 4.6 per cent, was less than that for staff or field officers and practically the same as that for non-commissioned officers and enlisted men, 4.5 per cent. It is probable that during all ages there has been a tendency to protect the king or leader in battle as well as the princes and nobles and also the standard.

I know of no statistical proof on this point but it seems reasonable to suppose that such would be the case. There are not many instances—in medieval and modern times—of kings killed in battle; Richard III of England was, and also Gustavus Adolphus, and Charles XII of Sweden, and James IV of Scotland. James II of Scotland was accidentally killed at the siege of Roxburgh 1460, by the bursting of one of his own cannon. Artillery was a novelty and James, whose curiosity was great, was standing too close. There are doubtless other instances of sovereigns killed in battle that I do not recall, but I feel rather sure that the actual total would not equal the approximate thirteen per cent for German officers of all sorts, and enlisted men killed in the recent war. There are 354 sovereigns in my studies[19] of the capacities of rulers and conditions of prosperity of the fourteen countries over which they ruled. To be thirteen per cent, the number of kings killed would have to be forty-six instead of five. I do not believe I have forgotten such a difference. The importance of the matter is not commensurate with the small statistical array here given for royal deaths in battle; but it is very suggestive that there are now, and have been in the past, certain eugenic forces in warfare which have preserved the higher pedigreed and socially superior strains. These, if gifted at all, have nearly always been especially gifted in the arts of war and government. We see here a natural selection tending to preserve both the artist and his art. Another point in confirmation is that the latest Great War took apparently only two princes of the strictly royal sovereign families from a total population of approximately 1,750, —a striking contrast to Germany's losses for officers and men which we remember was thirteen per cent! If princes and nobles are thus protected from proportionate death-loss to such a very marked degree, it is not too much to suppose that the social classes ranking close to royalty are also somewhat immune to the lethal effect of war. As far as it goes it obviously is another factor preserving aristocracies and perpetuating a class of more than average intelligence. Crucial evidence of this last statement regarding their intelligence is given especially in my *Influence of Monarchs*.[19]

War Years Not Lessening

For many centuries, one half the time has been devoted to war and one half to peace. Man seems to have two natures,—equally normal yet diametrically different. In theory and in real desire most men, in times of peace, are pacifists, but when the herd spirit of war is on and patriotism is on high, such theories and such instincts are supplanted by other instincts and other ideals equally fundamental and equally valuable in the preservation of races. The warring spirit has nothing to do with any free-fighting spirit, or with single-handed fights, or duals. Its origin is entirely different, and also its fate. I brought out in 1915[29] these points, which appear still to be almost entirely overlooked, and I then furnished—from the history of mediaeval and modern Europe—the statistical evidence supporting my position.* As there does not appear to be any likelihood of war soon becoming less formidable

*Neither England nor France, from the eleventh century to A. D. 1900 show any diminution of war years. The earlier half is the same as the latter, 50% war and 50% peace.

or less frequent, the question of a selective death-rate favoring the higher social classes is an important one.

The fact that thoroughbreds (or more nearly thoroughbreds) give expectation of greater net fecundity than crossbreeds is merely another link in the theory of social conification which means a relatively smaller quantity at the top, but a finer quality for this elite. In other places I have shown how aristocracies by force of natural and social selection, form, improve, and persist. Here is presented further evidence of the antiquity of the chief families of Continental Europe. The antiquity of most British peerages was demonstrated in the JOURNAL OF HEREDITY, September, 1928. The greater net fecundity of superior individuals over inferior members of the same families was proved in 1903 for royalty and 1926-7 for Harvard graduates,—confirmed by J. C. Phillips and others.[9] Therefore there is now much evidence from many directions pointing more optimistically than was formerly supposed towards a mental and moral evolution among the *higher social classes* which are being apparently separated biologically more and more from the dangerously fecund proletariat.

Postscript—Can We Say Why?

The Editor of the JOURNAL having written me that it would be desirable to know why the non-noble marriages are so much less fecund than the thoroughbred, I have added the following postscript.

The question is undoubtedly a difficult one to answer, and I should never have undertaken it had I not been asked to do so. Now that it has been asked I can see that it is pertinent and clarifying, and that it is quite possible to get some notion as to the many *whys*.

For the twenty-odd cases of Italian princes marrying outside of the nobility, I have been able to collect a reasonable array of information. I have been living in Rome for the past eight years and now, since the Editor's letter, I have systematically asked questions of a number of friends in whose judgment one can place confidence and who are connected with, or related by birth to some of these families, and consequently are in a position to know. My report concerns only broad differences and easily established facts, such as the probable reasons for the marriages, wealth of brides and grooms, general traits,—mental, moral, and physical,—and whether or not the marriages have been considered successful and whether or not they were thought to be love matches or were made for money. Additional facts as to ages at the time of marriage, legal separation and divorces are usually printed in the *Almanach*.

It is a little difficult to decide just how to present this rather unusual and extremely personal and private batch of material so that it may serve the interests of science and at the same time conceal the names of the persons discussed, but it seems perhaps easiest and best to take it up as a series of "case studies" as a physician might say, and at the same time comment on various phases as we proceed.

Instead of following the order of my notebook, I will skip about as one instance may suggest a similar, or perhaps a contrasted case. The reader will soon see that diversity is the keynote if there be any. Perhaps in spite of this the histories may fall into types.

The first case that strikes my attention (*B*, p. 2) is that of an American girl who married a younger son. She was very wealthy. The marriage was, according to report, "certainly for money." He had been deeply in love with a young noblewoman from another city, but the parents would not consent. The international marriage proved childless, and some years later the couple was divorced. He married, in a few weeks, the same noblewoman, then in the thirties.

The second case (*D*, p. 5) is half similar. This was "probably for love, yes, certainly, as all of the family were opposed." She was an Italian widow,

aged 38. He was 37. They have one child. Here the "why" seems evident in the advanced ages.

The third (*K*, p. 11) is unusual as it is a union with a rich American and has presumably turned out well. The prince was, himself, rich. She is very well thought of and well received socially. They have two grown children. One is a talented artist.

The fourth (*L*, p. 12) is another American marriage, this time not so fortunate. The aunt who was very rich promised her niece an annual income, but paid one year only. The niece and her husband took the case to court, but the aunt won, as it was "only a promise." There are two children. The husband is moderately wealthy.

It has been suggested by the Editor of the JOURNAL that perhaps those who make outside marriages are reduced in income with that loss of title and family standing that I have referred to as "demotion in rank," and that this is a reason for their smaller families. Such may be true in extreme cases among the higher sovereign houses. Among the Italians there is little or nothing of the sort. There is no loss of social caste. The idea that plenty of money favors plenty of children is very widely held. Personally I doubt its being more than an extremely minor factor. Let us take all the cases where the Italians have married wealth and contrast these with the others.

"B, p. 2" and *"K*, p. 11" have already been cited. They total one son and one daughter. Other instances of marriages with wealth are *"A*, p. 1," rich, no children; *"C*, p. 4," rich Jewess, has two sons; *"H*, p. 10," very rich, from Chicago, two daughters; *"N*, p. 28," rich English girl born in Paris, no children; *"O*, p. 29," Italian, "ugly but rich," or, on another authority, "very rich," one son, two daughters; *"P*, p. 34," New York oil-fortune heiress, went recently to Mexico to obtain a divorce, no children.

"R, p. 36" is a rare case of happy marriage, considered "a very happy marriage." She is very well thought of, one son and three daughters, the record number of offspring for Italian non-noble and noble matrimonies.

"S, p. 37" is a sad case. The bride was 24, the beautiful and charming grand-daughter of an extremely wealthy American, said to have left a fortune of $50,000,000. All has been lost or wasted. Now she is penniless and unbefriended. There are two children.

"T, p. 16" is a union with a rich Jewess now bearing one of the historic names. They have one son and one daughter. The prince, though not brilliant, is considered a fine character. In the next, *"V*, p. 18," the prince was himself moderately rich. The marriage was "probably for money." They have been married six years and have one child.

"V, p. 22" is another case of an unsuccessful marriage with a rich American from whom he has separated. Lastly we have *"W*, p. 20," a rich widow born in Kentucky. The records disagree as to her age at the time of the marriage, one giving it 44 and one 47. There appears to be one child.

We have now reviewed fourteen cases where wealth abounded. The total is only eighteen offspring, or an average of 1.3, which is a great contrast with the thoroughbred's average of 2.9. This shows that it cannot be a question of financial support.

There are seven cases where the wives were distinctly poor. Here there are eight offspring. Apparently it makes little difference one way or another whether the wives are rich or poor. As a statistical sum seven is certainly a very small number, yet in this instance, combined with the foregoing fourteen, it shows one thing clearly and refutes a good deal of the nonsense of attempted generalization. It proves that the reasons for wedlock among these people are as diverse and complex as can well be found anywhere. Let us review the "dotless" marriages.

The first case, *"D*, p. 5" has already been cited. He was 37 (without much money), she 38, a widow—an Italian, without any money. She is well spoken

of,—a "bella donna," which in Italian means more than merely beautiful. The prince's family were all opposed. It is considered a love marriage. There is one daughter, 18 years old.

The next one on the list (one of the grandest of princes) married a midwife of low social position, was divorced from her, and married another woman of similar sort (*E*, p. 6). He was 35 at the time of his first marriage and nobody knows how old the woman was as this almost universally rendered bit of information is, in her case, not given in the official *Almanach*.

"*F*, p. 3" is of a very rare type among non-noble marriages. They were both young; she without fortune, and he not more than moderately rich. They have four children, which is the high total for Italian misalliances. The marriage was "presumably for love," yet ended in a legal separation ten years later.

"*G*, p. 8" seems to be a case of a good-looking, large-sized, determined widow "getting hold" of an eligible middle-aged bachelor, not rich but an advantageous match for her from the social point of view. She was without any property; he with little. She is considered "vulgar" and "not received in the inner circle." They have been married a number of years,—no child. People speak of her as "an adventuress."

"*L*, p. 12" has already been described. It is curious as the expected dot did not arrive. There are two children.

In the next case (*M*, p. 17) we have a story entirely different from any of the others. A prince who was very much of an invalid married his nurse. Both were somewhat past the average age of marriage. There is one child.

The last case on the list does not speak favorably for misalliances. It tells of a rich and mature prince of historic name who married a young American divorcee, "beautiful but always a tremendous drinker," with a character not highly praised. The marriage can hardly be called successful. There are three cases about which no private information has been obtained, but their omission can be of little significance. There are also eight more examples of Italian princes marrying non-nobles, but these all live outside of Rome and therefore are not included in this particular analysis.

There are two cases outside of Italy that I can add from personal and private information, and are worth including. The first is clearly a case of sex-appeal predominating. The prince, himself wealthy, married first a very handsome young widow who cared little for him. They were divorced and he then married his former mistress, who was always socially déclasse. He was a kind-hearted, generous man who cared more for his own pleasures than for the approval of his family. The other case is typical of just the opposite sort of marriage. Here it was clearly a prince who married for money and a charming, beautiful American girl who was foolishly captivated by a famous title. The prince himself was far from attractive, and morally his rating is definitely established, inasmuch as after spending his wife's money he was convicted and imprisoned for fraud! All the fortune disappeared and she died poor. She was exceedingly vivacious, witty, and generous to a fault. There was one child born about a year after the marriage. After that no more.

In generalizing on these 24 cases one fact is incontestable. There are no instances of those large families of five or more children which are so frequent among inbred European aristocracy and so rare among the upper social classes in the United States. Another point seems to be that they can be readily divided into two categories. Either the wedding dots are considerable or none at all. When they marry for money they seldom marry also for love. The evidence is that these persons are usually not of the domestic or children-loving type. When they marry on account of sex-appeal, or are entrapped by the impecunious but ambitious social climber the same generalization holds.

Divorces and legal separations are frequent. Here there are eight cases. To this may be added that the parties concerned average about 32 years at the time of wedlock instead of about 26, as among pure-breds. It cannot be doubted that the desire for parenthood causes early marriages rather than late.

The reasons why cross-breds have fewer children are seen to be complicated. It is sometimes one reason, sometimes another. It is not a question of money or lack of money. Everything considered (their advanced ages at marriage, their frequent divorces and separations) would lead to the idea that the chief reason why they have few children is because they do not especially want them. Those who marry within their own circle evidently do want them,—averaging about three. Since the parental instinct is doubtless inherited this tends to preserve the pure aristocratic strains. In such ways as this nature is constantly pulled toward the normal.

Literature Cited

1. DE CANDOLLE, A. Histoire des sciences et des savants depuis deux siecles. Geneve, etc. (H. George), 1873. Contains lists of scientific names objectively (impersonally) compiled.
2. COX, C. *Genetic Studies of Genius.* Vol. II. 1927.
3. ELLIS, HAVELOCK. A Study of British Genius. London: Hurst and Blackett. 1904. Also 2nd Edition. A valuable study based upon the *Dictionary of National Biography.* Contains lists of British men of genius and talent, objectively derived and useful for further study.
4. GALTON, FRANCIS. Hereditary Genius. An inquiry into its Laws and Consequences. 2nd Ed. London, Macmillan. 1892. The earliest of biographical statistical studies, first published in 1869. Many of the lists of names are not compiled by any strictly objective method. The part played by environment is undeterminable.
5. ————. English Men of Science, Their Nature and Nurture. London, Macmillan, 1874. Fellows of the Royal Society.
6. ————. Noteworthy Families. London, Murray. 1906. Families of scientific men objectively studied.
7. IRELAND, ALLEYN. Democracy and the Human Equation. 1921.
8. ODIN, A. Genese des grandes hommes, gens de lettres francais modernes. 2 vols. Paris: H. Welter. 1895. A study of 6,382 French men of letters. Valuable for its facts. The conclusions regarding heredity *vs.* environment are unwarranted.
9. PHILLIPS, J. C. *Harvard Graduates Magazine,* June, 1927.
10. POPENOE AND JOHNSON. Applied Eugenics. 1933.
11. SALLER, K. *Einfuhrung in die menschliche Erblichkeitslchre und Eugenik,* Berlin. 1932.
12. STODDARD, L. The Revolt Against Civilization. New York: Scribner's. 1922.
13. TERMAN, L. M. Genetic Studies of Genius. Vols. I and II, Stanford University, 1925-27.
14. WIGGAM, A. E. Fruit of the Family Tree. Indianapolis: Dobbs Merrill Co.
15. ————. The Next Age of Man. 1927.
16. WOODS, F. A. The Correlation between Mental and Moral Qualities. *Pop. Sci. Month.* 63: 516-525. 1903.
17. ————. Mental and Moral Heredity in Royalty. New York: H. Holt. 1906.
18. ————. Historiometry as an Exact Science. *Sci. N. S.* 33: 568-574. 1911. (Rep. in Appendix in "The Influence of Monarchs," 1913.)
19. ————. The Influence of Monarchs. New York: Macmillan, 1913.
20. ————. Heredity and the Hall of Fame. *Pop. Sci. Month.* 84: 445-452. 1913.
21. ————. Good Qualities are Correlated. *Jour. of Hered.* 10: 84-86. 1919.
22. ————. The Conification of Social Groups: Evidence from New England Families. Eugenics, Genetics and the Family. *Sci. Papers of the Sec. Int. Cong. of Eugenics.* Pp. 312-328. Baltimore: Wilkins. 1923.
23. ———— The Survival of Ability. *Science.* 66: 429-430. 1927.
24. ————. Is the Human Mind Still Evolving? *Jour. of Hered.* 18: 305-308. 1927.
25. ————. Successful Men Have Larger Families. *Jour of Hered.* 19: 271-279. 1928.
26. ————. Mental Evolution and Social Conification. *Metron* (International Review of Statistics) x 7: 121-142. 1928.
27. ————. The Perpetuation of Old Families — Inheritance of Parental Instincts and "Social Conification." *Jour. of Hered.* 19: 387-398. 1928.
28. ————. The Relation Between Intelligence and Birthrate. Comitato Italiano per lo studio dei problemi della popolazione. (Publication of the International Congress of Studies Regarding Population Problems.) Rome, 1931.
29. WOODS, F. A., AND ALEXANDER BALTZLY. Is War Diminishing? Boston and New York: Houghton, Mifflin and Co. 1915.

NEW FILBERT HYBRIDS

C. A. Reed

Bureau of Plant Industry, United States Department of Agriculture

AN entirely new hybrid form of filbert, which appears not unlikely to take an important part in future nut growing of the Eastern States, has been produced by artificially crossing certain varieties of one of the eastern native hazels, *Corylus americana* Marsh, with some of the leading commercial varieties of the European filbert. From about 4,000 hybrid trees grown during the last 18 years approximately one-half have been brought into fruit. Fully 25 per cent of the latter have been definitely discarded, and of the remaining 1,500 trees two have been given varietal names and are now being disseminated to a limited extent. About 50 others of much promise are under close observation. With few exceptions, bearing plants of this new form are of the F_1 generation, and some are of such high merit that in the open market they should compete advantageously with the average European varieties now grown or sold in this country.

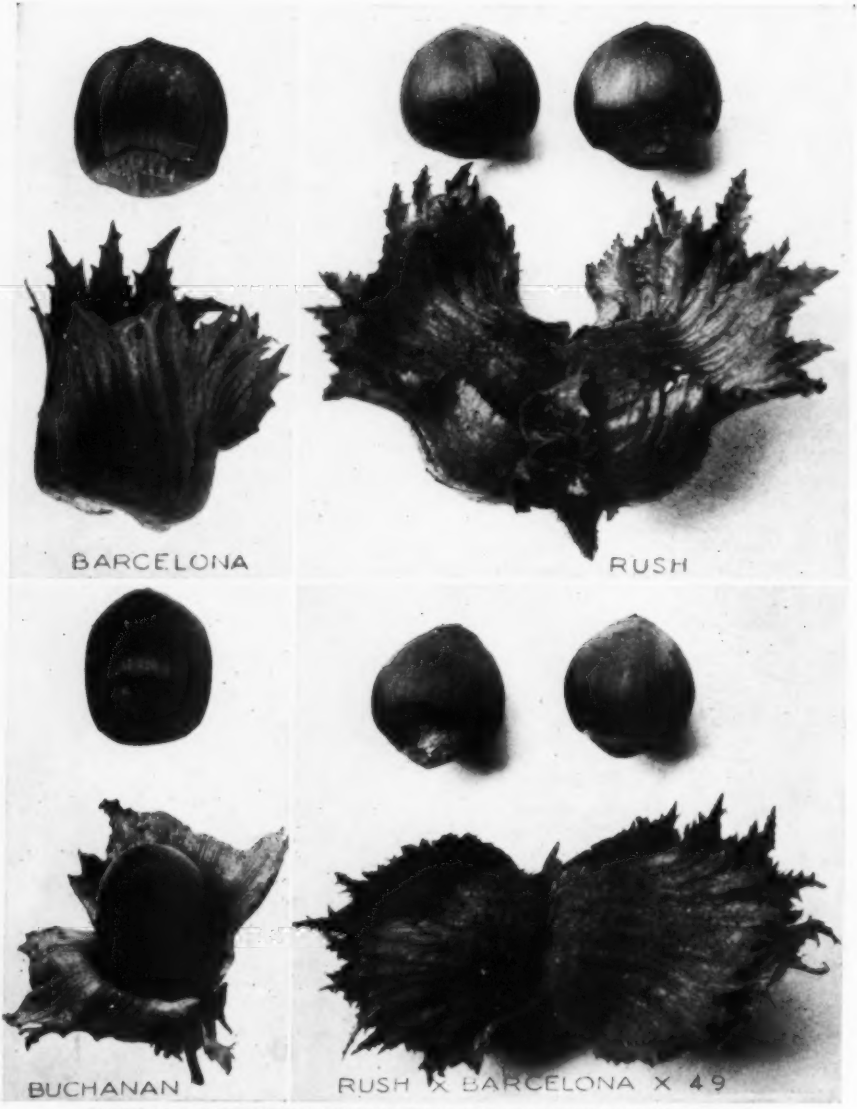
A *Mayflower* Plant Immigrant

Filberts from Europe were probably introduced into the United States by the first settlers. Additional introductions have been made from time to time, but no varieties have become successful commercially in the East. Plantings are known to have been made in years past from lower New England south to Georgia and west throughout much of the Mississippi Valley. Frequently northern plantings have given temporary promise of commercial possibilities, but as a rule all have perished within a few years. Usually the flowers were destroyed by severe temperatures, or the plants were killed by a fungus disease apparently prevalent on the native hazel everywhere that species was at all abundant, and which spread quickly to the filbert when brought into the neighborhood. Farther South, filbert production has been practically nil, due to the inability of the plants to adjust themselves and their blossoming habits to mild and variable winters.

The new hybrid filberts have so far been developed largely by the late J. F. Jones of Lancaster, Pa., and the Bureau of Plant Industry, working separately. Plants from the Jones stock are under test mainly on the home grounds of Mr. Jones' estate and on those of Mr. Harry R. Weber near Rockport, Ind. The trees of the Bureau of Plant Industry are growing on grounds of the U. S. Horticultural Field Station near Beltsville, Md., and at the New York State Agricultural Experiment Station at Geneva. Recently similar breeding experiments have been conducted by the Department of Pomology at Cornell University, Ithaca, N. Y., and the New York Agricultural Experiment Station at Geneva. Also working more or less independently, S. H. Graham of Ithaca, N. Y., has brought into bearing a number of F_2 plants from the Jones stock, several of which appear particularly promising.

The hybridization of hazels and filberts was begun in 1917 by Mr. Jones after he had made a diligent study of the filbert in the East. He was convinced that except under the most highly favorable conditions there was no likelihood of commercial development of European varieties in any part of the United States except on the Pacific Coast.

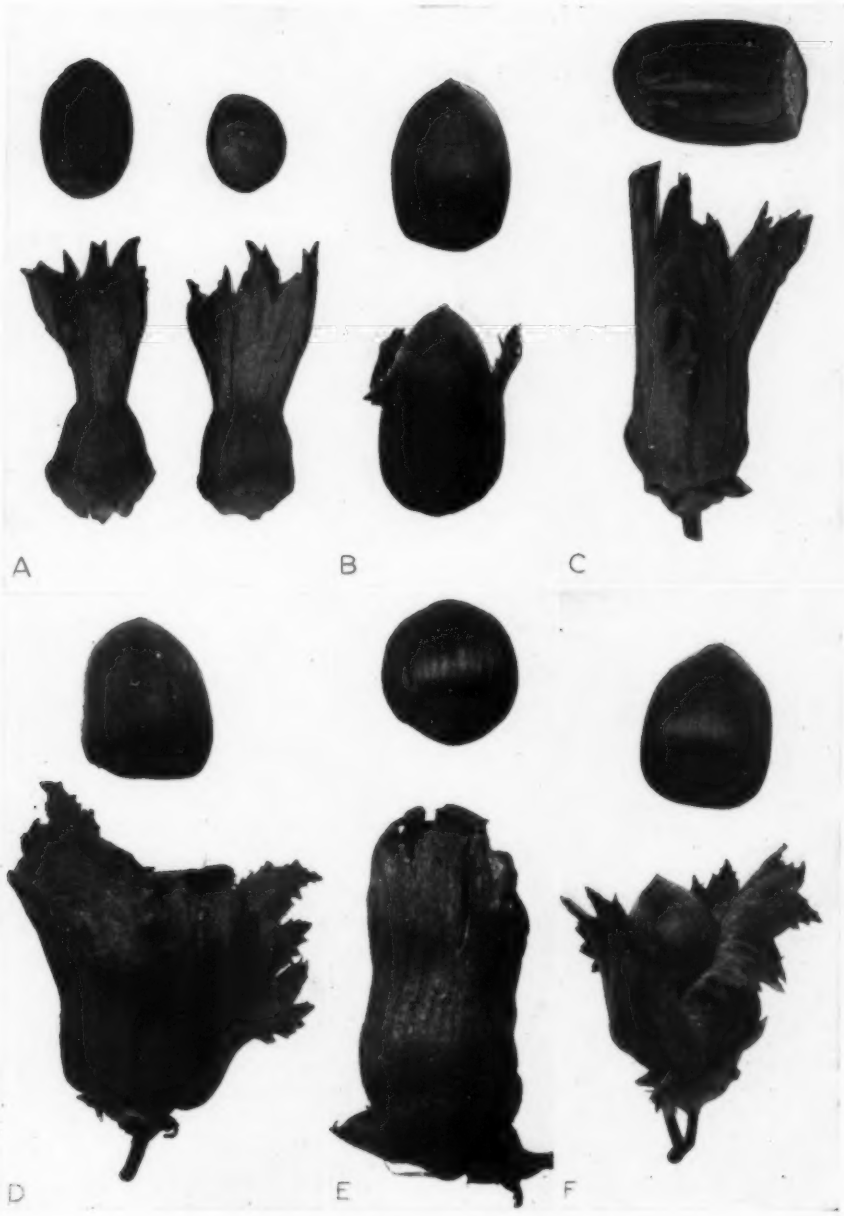
For two years Mr. Jones made crosses of European filberts with pollen of the native Rush variety of hazel, but failed to procure a single nut this way. In 1919 he reversed the order by applying European filbert pollen to the pistillate flowers of the Rush variety of hazel, with highly gratifying results.



HYBRIDS BETWEEN EUROPEAN AND AMERICAN FILBERTS

Figure 3

The Rush is a variety of the American filbert, and the Barcelona is of European origin. The Rush was used as the pollen parent in all crosses made by the late J. F. Jones which produced superior hybrids. The Buchanan, the most promising of the Jones hybrids, is of Rush × Barcelona parentage. The other Rush hybrid here shown is a selection made by the U. S. Department of Agriculture.



PARENT VARIETIES AND HYBRIDS

Figure 4

Above are shown three European varieties of filberts: *A*, White Aveline; *B*, Italian Red; and *C*, Du Chilly. Below are shown three hybrids from these varieties: *D* is a Rush × Du Chilly hybrid, *E* is a Rush × White Aveline hybrid and *F* is the named variety Bixey, originated by the late J. F. Jones. It is a hybrid between Rush × Italian Red.

By 1924 fully 200 seedlings out of the first crosses were in fruit and by 1928* about 750 had been grown by him.

A single pistillate parent, the Rush hazel, was used in all crosses made by Mr. Jones. It had originated as a roadside stool in the southern part of Lancaster County and was discovered about 1900 by the late J. G. Rush of West Willow. Plants from the original stool taken by Mr. Rush to the rich soil of his garden soon attained heights of from 15 to 20 feet and bore heavy crops. However, Rush nuts are entirely too small and the kernels of too poor a flavor for the variety to be of commercial value.

Origin of Bixby and Buchanan Varieties

The pollen parents used included Barcelona, Cosford, Daviana, Du Chilly, Italian Red, and probably others. The individual hybrid plant which impressed Mr. Jones most favorably was Number 200, a Rush × Italian Red cross. This plant bore nuts of considerable merit for a number of years. About 1930 it became so crowded by other plants that both the yields and character of the nuts were uncertain and its recognition as a variety was temporarily withheld. Its neighbors were then removed and in due time both the nuts and the yield again became normal. In 1934 this hybrid was given the varietal name of Bixby after the late Willard G. Bixby of Baldwin, L. I., a friend of and coworker with Mr. Jones. (See Figure 4*F*.)

While recognition of hybrid Number 200 was being withheld, Number 92, a Rush × Barcelona hybrid, attracted attention because of the general high character of the nuts and the heavy yields produced. This plant attained varietal status in 1931 when it was named Buchanan after President James Buchanan, who spent his last years in Lancaster and was buried in a local cemetery. (See Figure 3.)

Filbert breeding in the East was actively undertaken by the Bureau of Plant Industry in 1927, when approximately 2,500 crosses were made between leading varieties of filbert then just beginning to bear in the Bixby orchards near the southern shore of Long Island. At that time, a number of European varieties were thought to offer considerable promise of hardiness in at least certain favorable localities of the East, including in particular that part of Long Island. All crosses, therefore, were confined to these varieties. The results were exceedingly disappointing, as severe weather killed all but a very few of the cross-pollinated flowers. The nuts that matured were planted and about 50 plants are now growing at Beltsville, Md., but these appear to offer no greater promise of hardiness or value in other ways than do the standard varieties growing on the same grounds.

Beginning in 1928 and continuing annually for five years, large numbers of crosses were made in the Bixby orchards, mainly between the Rush hazel as the pistillate parent and such European varieties as Bolwyller, Kentish Cob, Red Aveline, White Aveline, Red Lambert, White Lambert, Imperial and Brixnut, in addition to all those previously used by Mr. Jones. To some extent, the Littlepage variety of hazel was used as a pistillate parent, and such species as *Corylus colurna* L., *C. maxima* Mill., and *C. heterophylla* Fisch., were chosen as the pollen parents.

There is much uncertainty as to the true species parentage of many of the European filbert varieties. Apparently some are of pure *Corylus avellana* stock, some are from *C. maxima*, and others are hybrids between these two. The possibility of tracing the exact parentage in all cases appears remote. In this breeding work, such crosses as were made with pollen of other distinct species were for the purpose of ascertaining the extent to which hybridization would be possible rather than with

*Mr. Jones' death occurred in January, 1928.

the expectation of developing superior varieties.

Dominance in Crosses

In connection with these hybrids, it is interesting to note the extent to which certain characteristics of the pollen parents appear to be dominant. The hybrid plants seem to be much more hardy than the pollen parents. They are also less inclined to throw out suckers, more erect in habit of growth, more vigorous, and more easily trained to tree form than are the pistillate parents. The foliage of the trees is invariably much like that of the pollen parents in that the leaves are rather thick, dark, and stiff as compared with those of the Rush variety. Many of the young plants are particularly handsome, being erect, more or less goblet-shaped, symmetrical and compact. They tend to retain their foliage until frosts occur in October or November. In size, form and color of nuts, thinness of shell, plumpness and appearance of kernel, the nuts are much like those of the pollen parents. In quality and flavor, the hybrid kernels are often distinctly agreeable.

The *colurna* crosses were of course the most violent, as that is typically a tree-growing species, and as expected, the number of nuts obtained was exceedingly small. Very few of the resulting trees show positive *colurna* characteristics, although in a few cases the color and general character of the bark is distinctly like that of the pollen parent. The few nuts thus far obtained from these hybrids have largely been without kernels, and the total number entirely too limited to justify conclusions as to possible merit.

An undesirable characteristic of the Rush hazel is an evident inability to endure training to a single stem. In the Bixby planting of about 15 Rush trees, no less than three broke off below the ground and blew over when 8 to 12 years of age. The roots of the Rush trees are highly persistent in their habit of suckering and apparently are incapable of developing sufficient strength under ground to sustain the plant when the part above ground is forced into a single stem. Of all the hybrid plants yet grown, the great majority observed by or reported to the writer are yielding satisfactorily to the single stem method of training.

The purpose of reporting these hybrids at this time is chiefly that of record. Obviously, with the exception of Buchanan and Bixby which are in commercial hands, no varieties are likely to be announced or available for trial planting until their value has been demonstrated. In addition to determining the merits of the nuts and the performance of the trees, it will be necessary to ascertain the extent to which these hybrids are dependent upon others for pollen. Whether they will be largely self-sterile like the pollen parents in this respect is yet to be determined.

Incidentally, a chlorosis, or leaf-burn of undetermined cause, has made its annual appearance on many of the trees. Very few trees at Beltsville are entirely free from this disorder. In severity, the range is from faint traces to nearly 100 per cent destruction of foliage by the end of August, or at about the time the nuts begin to ripen and fall to the ground. It is rare that the kernels of the nuts are well filled when the foliage of the trees has been badly infected.

HAPLOID AND NORMAL MARGLOBE TOMATOES

Figure 5

The haploid tomato, having in its cells only a single chromosome of each of the set of 12, is much smaller and less vigorous than the normal diploid form. The size of the fruit is further reduced by the fact that very few seeds develop. The rare seeds in haploid fruits are the result of self-fertilization and are necessarily homozygous (pure) for all genetic characters, as explained in Figure 7. Thus these rather unpromisingly small fruits may be of considerable importance to the plant breeder because they offer the quickest way to obtain pure lines so essential to produce uniform varieties. (See cover illustration for a photograph of the entire fruits.)

A HAPLOID MARGLOBE TOMATO

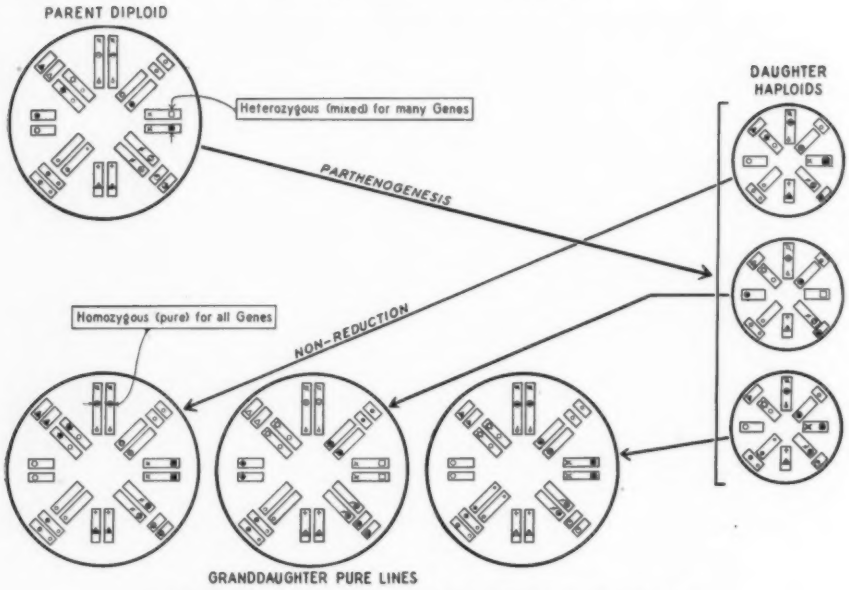
Practical Application of a "Short Cut" for Making Pure Lines

HAPLOID AND NORMAL TOMATO PLANTS

Figure 6

A haploid Globe tomato plant between two normal sibs, showing characteristic dwarf habit of growth. Such plants, containing in each cell only a single set of twelve chromosomes, instead of the normal duplex complement of 24 chromosomes, are found very rarely in large field cultures (one plant in 50,000-100,000). These rather weak-growing dwarfs are of great theoretical interest, and of no little practical importance because from them can be obtained by self pollination, or by vegetative growth from callus tissue, genetically pure lines "at one jump," instead of by generations of inbreeding. (See Figure 7.)

AN example of how discoveries in pure science may have very considerable (and often unexpected) practical importance is contained in the photographs reproduced herewith of a haploid of Globe and Marglobe tomatoes, and of "pure line diploids" derived from the latter. The first haploid plant to be recognized was reported in the Jimson weed by Blakeslee and Bergner in 1923. One of the first illustrations of this haploid Datura, and possibly the first photograph of a haploid plant to be published, appeared in this Journal in 1924. In this article Blakeslee pointed out the possible value of such plants, whose cells contain only a single set of chromosomes, as a short cut in the production of pure lines. The chart Blakeslee published at that time is reprinted herewith with additions, as it gives in very concise form the theory of producing pure lines by the "haploid method." Since a haploid plant contains only one set of chromosomes the doubling of this single chromosome set automatically produces a plant which is homozygous for every gene. In practice such a chromosome doubling has been brought about in two ways: by the occasional self-fertilization of rarely-occurring unreduced gametes and by the so-called "decapitation callus" method. This method grew out of Winkler's work on producing graft-hybrids by cutting through a graft union. Tissue arising at the point of junction of stock and cion was sometimes chimeral in nature, and Strasburger suggested that some of these "graft-hybrid" single cells might contain chromosomes from both stock tissue and cion tissue. Lindstrom and others showed that in the case of haploids Strasburger's suggestion was confirmed, for considerable numbers of diploid shoots were produced from the callus tissue of decapitated haploid plants.



PRODUCTION OF PURE LINES FROM A HAPLOID

Figure 7

This chart shows how in the third generation from a heterozygous (genetically impure) plant a pure line may be produced. By parthenogenesis of a normal egg cell a plant with but a single set of chromosomes arises,—a haploid. Occasional self-fertilization of haploid non-reduced eggs and pollen produces a plant which necessarily inherits the same characteristics from both "maternal" and "paternal" germ cells. Thus it is genetically uniform.

Such diploid shoots are "pure lines," being necessarily homozygous (barring mutations) for all genes.

Such absolutely uniform genetic material has obvious possibilities in maintaining and multiplying a superior variety. In hybridization and selection the value of such genetically uniform strains may also be very great, since the selfed seed from such a homozygous plant would be exactly like the parent form, and since crosses between two such pure strains would always be (at least until mutation within the pure line changed the picture) of identical genetic constitution.

What is perhaps the first use in practical plant breeding of these developments in theoretical genetics is illustrated in the accompanying photographs, furnished through the courtesy of Mr. Gordon Morrison, of the Ferry-Morse Seed Breeding Institute at Rochester, Michigan. Mr. Morrison has used a haploid plant of the Marglobe Tomato, which appeared in the test cultures of the Ferry-Morse Seed Breeding Institute, to produce an absolutely uniform strain of this valuable variety. The Marglobe tomato was originated by the U. S. Department of Agriculture and introduced in 1925 as a wilt resistant form adapted for growing in the Southern States, where that disease is a serious problem. The variety was almost immediately accorded wide recognition as an outstanding general purpose variety, and is extensively grown today. The way in which the haploid "mutant" has been used to maintain a uniform strain is explained by Mr. Morrison in the letter accompanying the photographs:

We note your interest in the first paragraph of our letter. The production of this pure line of Marglobe Tomato is of considerable practical as well as theoretical interest to us. We believe it is the first occasion of the pro-

duction of a pure line of seed propagated material by means of a haploid plant. The pure line was not produced by the decapitation callus method, as you seem to suspect.

The haploid plant of Marglobe was discovered in a breeding culture growing on the Oakview station of the Ferry-Morse Seed Breeding Institute in 1929. It doubtless arose due to parthenogenetic development of a reduced egg and the ten seeds which it bore were doubtless each due to the chance union of rare normal egg and pollen grains which were the result of non-reduction.

The haploid plant met all the criteria by which these are commonly judged; namely, the plant was miniature in size in all respects, more than 99 per cent of the pollen was shriveled, and most of the flowers fell off. We did not think it was necessary to make chromosome counts particularly since the writer had had considerable experience with haploids while associated with Dr. A. F. Blakeslee in the Genetics Department of the Carnegie Institution of Washington. If it was a haploid as it surely appeared to be, the offspring should be normal and exactly similar to one another. This proved to be the case and the ten normal offspring were ideal specimens of this splendid variety of tomato. The pure line thus produced has been increased under our own observation to more than 250,000 plants with no variation whatever of significance to plant breeders. There have been three suspected haploids which were not examined further since our pure line was well established and entirely satisfactory, and one plant in the second normal generation gave rise to progeny with slightly smaller appearing fruits but this was not different in any other respect, and the slightly smaller size of fruits may have been due to environment. Thus we feel justified in saying that we have an absolutely pure line of Marglobe and this is what the trade is using at Supreme Marglobe.

We enclose two prints which illustrate a fruit from the haploid plant compared with a fruit taken from a normal sib. You will note the similarity in form and the difference in size. In the cut fruit you will note that the fruit from the haploid shows one seed imbedded in the flesh in the lower side of the right half. The 250,000 normal plants in the generations propagated all bore fruits exactly like the normal fruit illustrated here.

Mr. Morrison has elsewhere reported (*Proc. Sixth International Genetics Congress*, Page 127) the occurrence of additional haploids in this and other varieties. In all three haploids have appeared in the Marglobe, two in the Gulf State Market, one in Globe, and one in Earliana. As is to be expected, the Marglobe haploids are not exactly alike, and it may well be that the diploid progeny of one such sister haploid may be superior to that of another. The extent of variability in haploid-produced pure lines from a given variety would depend on the extent to which the parent form was homozygous. From a highly selected, uniform variety like the Marglobe, the haploid progeny, and the "haploid-produced pure lines" would be expected to be much less variable than similar pure lines produced by an array of haploids from the F_1 generation of a cross. Haplois from highly heterozygous material would give new pure breeding forms much more quickly than by ordinary methods of crossing and selection.

Thus there is a possibility that Mr. Morrison's pioneer breeding work with haploid tomatoes will some day be cited as the beginning of "haploid plant breeding stations" where this interesting technique will be utilized to the limit of its possibilities. Instead of growing acres of seedlings and throwing all but a handful away, there may come a time when our future Burbanks will grow acres of haploids to get a few dozen seeds—each one of these representing the beginning of a unique pure line, which will form the basis of later breeding experiments.

At the moment we do not know what the result might be of cross pollinating haploids, so that the occasional viable pollen of one plant would fertilize the unreduced egg of a haploid of another parentage. Theories of heterosis now the subject of controversy differ in accounting for the part of hybrid vigor. Depending on which view we accept, we might be tempted to predict either that such plants would show considerable hybrid vigor or none at all. When such an experiment is tried it might have considerable importance in settling certain moot points concerning the causes of hybrid vigor, and it might be very important in future breeding programs. Other "perhaps" uses of haploids might be suggested, so purely speculative, however, as hardly to be suitable subjects for plant patents—basic or otherwise.

R. C. C.

AN INHERITED TREMOR IN PEROMYSCUS*

R. R. Huestis and Elizabeth Barto†

IN the spring of 1934 the junior author discovered in a brood of three mice about three weeks old a young undersized Peromyscus which had a very pronounced tremor. The other members of the brood were normal in appearance and size for age. The affected mouse was observed subsequently to still exhibit the tremor and shortly thereafter it died.

The mother and father of the affected individual were members of the fifth and third hybrid generation, respectively, of young produced by mating *Peromyscus maniculatus gambeli* from Silver Lake, Oregon, with *P.m. rubidus* from North Spit, on Coos Bay, Oregon. These parents were related mice which had been mated for the purpose of investigating a white blaze on the nose.

The pedigrees of this pair and of all other parents of young mice with a tremor have two ancestors in common; *P.m. gambeli* SL ♀ 4 and *P.m. rubidus* CS ♂ 1. Either of these mice may have been the original carrier and we have presumably bred other potential parents of affected young for some time for the character is one that could escape detection, since it is not obvious at the age young mice are first observed and recorded, and affected young are usually dead by the time the brood is old enough to be removed from the mother for later record and marking. The blaze stock from which we have obtained all the young which had a tremor has had a high death rate in captivity and has not been particularly fertile. It has, however, been maintained for over two years, during which time eight females and two males have produced affected young. (Table I.)

Description of the Character

Mice with a tremor have been identified as early as two weeks of age. At this time the affected individual is not noticeably smaller than normal sibs and observed specimens did not shake while they were nursing. If detached from the mother or removed from the nest an affected mouse may exhibit enough tremor, when it attempts to walk, to separate it from normal sibs even though, at the time, the movements of all the young are poorly coordinated. As the mouse matures the tremor appears to increase somewhat in intensity and the young mouse fails to grow normally. The disparity in size between affected and unaffected individuals becomes the more pronounced the older the brood becomes (See Figure 3*B*).

As in case of heredity tremor in fowls, there is considerable variability in the intensity of the tremor. Some young mice shake with such rapidity that one almost expects to hear them buzz. (Figure 8*A*). Such individuals do not appear to have any rest from this shaking while under observation (and perhaps excited by this circumstance), although they shake with less violence when they do not attempt to walk. Other affected young can remain still (Figure 8*B*) but will shake as soon as they attempt ordinary movement. Besides the tremor which appears to affect both body and limbs the gait may be shambling and progress irregular, often with the body poorly supported. An affected mouse may run into obstructions as it progresses or fall and lie sprawled in an unnatural attitude.

Young mice with a tremor may be

*This research has been assisted by the National Research Council and the Research Committee of the State System of Higher Education.

†University of Oregon, Eugene, and Cranbrook Institute of Science, Bloomfield Hills, Michigan, respectively.

TREMBLING DEER MICE

Figure 8

The upper photograph shows a three weeks old Peromyscus with pronounced tremor attempting grooming response. Taken at 1/50 of a second. It may be seen by the immediate background that the mouse is in focus. Thus the blurred outline of the mouse is due to his rapid motion.

Below are an affected (left) and unaffected Peromyscus from the same litter taken at about four weeks of age to show size difference. The normal mouse is slightly out of focus. The affected mouse did not shake except when it attempted to move.

repeatedly found separated from the remainder of the brood. While this suggests abnormal behavior, since the normal members of a brood stay together, this behavior may be restricted to times when the observer approaches a nest box. Affected animals can hear. A sharp noise which typically produces a muscular spasm in normal Peromyscus serves, in affected individuals, to momentarily check the tremor. We made no accurate determination of eyesight. Affected mice have been observed to carry out a rather brief grooming response. They appear to have some trouble in initiating this but, like the paralysis agitans patient who cleaned his teeth without effort, they need only make contact with their faces to bring about a thorough grooming of the point of contact!

All affected individuals have died before they were five weeks of age, most of them in the third or fourth week of life. Individuals separated with the mother have not survived any longer than those left with their sibs. As remarked previously the survival rate is poor in the stock carrying the tremor, but the unaffected relatives usually live a few months, and one heterozygous animal is now over two years of age. The fact that death has taken place at about the same period of life in all cases, together with the stunted and wizened appearance of the affected individual before death, makes it appear quite possible that the genes which produce the character can be considered lethal in effect. In an inherited tremor in chicks, which has a number of similarities with this condition in Peromyscus, Hutt and Child were able to raise one out of thirty-five individuals to sexual maturity. However, there is, in chicks, some lessening of the tremor as the animal gets older, a situation that we have observed no sign of in Peromyscus.

Inheritance

Since no mouse with a tremor has survived, all affected young have been produced by unaffected parents. These are referred to in Table II as carriers. As remarked previously, all such parents have been related mice. This suggests that the tremor is due to a recessive gene. The ratio of non-tremor to tremor departs somewhat from a normal 3:1 ratio but not seriously enough to refute the theory when the difference in viability of the two classes

of young is considered.

Parents of affected young have failed to produce affected young in a number of other matings. Most of these matings have been tests, within the same stock, for heterozygous individuals. They do not necessarily serve as a critical test of genotypes since it is possible for a parent to have several broods before a living affected young mouse is discovered. Mice recorded in the table as unidentified were those that died between the time a brood was first recorded and a later investigation was made and those which had a tremor identified.

Summary

1. A nervous condition has been discovered in the deer mouse characterized by trembling of the limbs and body and accompanied by deficient growth and, in the individuals so far noted, death before five weeks of age.

2. This syndrome appears to be due to a recessive mutation of a single gene locus, affected individuals all having been produced by related unaffected parents.

3. We have evidence that the disorder was introduced into our stock by one or the other of two trapped Peromyscus.

TABLE I. Females Mated with Carrier Males

Dam				Young		
			Broods	No Tremor	Tremor	Not Identified
F4	♀	90	2	6	2	
F5	♀	9	1	3	1	
F5	♀	41	3	9	1	
F5	♀	66	6	18	4	2
F5	♀	64	5	12	1	4
F6	♀	100	1	2	1	
F6	♀	241	1	3	1	
F7	♀	39	1	2	1	
			20	55	12	6

TABLE II.

Parents				Young		
				No Tremor	Tremor	Not Identified
Carriers	×	Carrier	obs.	55	12	6
			exp.	50	17	
Carrier	×	Others	obs.	85		9
			exp.	85		

Literature Cited

1. Huestis, R. R., and Elizabeth Barto. Recessive genes in geographic races of Peromyscus. *Am. Nat.*, 69, 65, 1935.

2. Hutt, F. B., and G. P. Child. Congenital tremor in young chicks. *Jour. of Hered.*, 25:341-350, 1934.

Parenthood Institute Popular

OVER six hundred persons attended the Thiel College two-day institute on Parenthood and Home Relations held November 5 and 6 at the college in Greenville, Penna. Planned by President Earl S. Rudisill, the institute surveyed its six sessions parent-child relationships from early childhood to college age. Speaking at each session was Dr. Gary C. Myers, noted child psychologist and newspaper columnist. His talks included: "Training Our Children for Marriage and Parenthood," "Our Children and Other People's Children," "High School Students and Their Parents," "College Students and Their Parents," "Children of the Same Family," and "Education in Responsibility."

THE BUDGERIGAR CULT

Love Bird Genes Furnish Sport(s) for Fanciers, Breeders and Geneticists

"BUDGERIGAR" to the cultists, but "Love Bird" to most of us in America, though if we are a little sophisticated we may refer to it as a Parrakeet. There are a number of species of love birds and parrakeets, but the budgerigar is a particular species (*Melopsittacus undulatus*) from Australia. If other names for it are desired, Dr. Russ is quoted as saying that it is "Variously known as the Undulated - Grass - Parrakeet, Budgerigar, Zebra-Parrakeet, Zebra-Grass-Parrakeet, Shell-Parrot, Scallop-Parrot, Warbling - Grass - Parrakeet, Undulated Parrot, Canary-Parrot, and Betcherrygah ('Good Bird') in the Liverpool plain and around Bigang in West Australia." Crew states that Budgerigar means "good food" in the dialect of the Australian aborigine; if so it would seem that the Australian black man must either be hard put to it for food or else have highly cultivated epicurean tastes. For the budgerigar is "one of the smallest parrots, being about the size of the sparrow, with long, pointed wings and tail, pleasing green and yellow plumage, on the upper surface partly dark and undulated, with beautiful blue spots at the upper side of the neck."

This little parrot first became known to science in 1805 and in 1840 the ornithologist Gould brought to England specimens which had been reared in captivity by his brother-in-law in Australia. These birds attracted favorable attention and further importations followed so that "soon every ship from Australia brought its quota, and quickly the budgerigar invaded North-west Europe, and for it a new era had dawned." In addition to its being an attractive cage pet, various color mutations or "sports" began to appear, which made it favorable material for the breeding of new varieties. It is of interest to note that all of these "sports" have arisen among the domesticated birds; only one color mutation, yellow, has been recorded in a wild population in its native home. A hint of the exquisite beauty of some of the color varieties that have been formed from the various mutations and their combinations may be gained from the names that have been applied to them, such as olive, skyblue, cobalt, mauve, in various shades; whites with suffusion of other colors; graywing and cinnamonwing; to name only a few of the possible combinations.

The ease with which the budgerigar can be reared as a cage bird, the relatively low expense because of its small size, and its great attractiveness of form and color, have led to budgerigar breeding becoming a popular hobby in the countries of Western Europe, particularly in England and Germany. Watmough indicates the growth of interest by citing that at the Crystal Palace Show in 1924 there was only one class for budgerigars, in which there were ten entries, while at the club show of the Budgerigar Society held at Olympia in 1934, only 10 years later, there were 1,257 entries. In the same year the membership of the Budgerigar Society had increased to nearly 2,000, which, however, represents only those keepers of budgerigars who are most interested in competitive breeding and exhibition of the birds. Such a wide spread of the hobby, resulting in a "cult" of the budgerigar, has naturally meant a corresponding interest manifest in magazines devoted to the "fancy" and to cage birds in particular, and as might be expected, to more pretentious books on the subject. Three such form the subject of the present review.

The first of these (though not in point of time), by Watmough,* is a general treatise, and may indeed be taken as an authoritative handbook on the whole subject. The author has not only drawn on his own wide experience but has made judicious use of the experience and writings of others. In addition to describing the color varieties and show standards he discusses their production and even enters into the genetic basis of color breeding, rather a hopeful innovation in a practical work. There follow chapters on aviaries and appliances, care and feeding, management, showing, judging—in fact, everything that would appear to be necessary to start a new enthusiast off in the game, and doubtless an equal number of valuable hints to the old timers. One can scarcely read the book without catching the contagion (psittaphilosis, *not* psittacosis) and rushing out to buy a cage and a pair of budgerigars, after which, one is presumably irretrievably lost in the hobby.

In view of the number of color mutations that have appeared in the budgerigar, including mosaics or "halfsiders," it makes excellent material for genetic research and it is not surprising that geneticists have availed themselves of the opportunity. The surprising thing, indeed, is that it has not been more widely used, particularly in this country.

The monograph by Steiner† is a typically German, thoroughgoing and scientific discussion of the subject to the time of its publication. It is directed primarily to the scientist, while the little volume on "The Genetics of the Budgerigar," by Crew and Lamy,‡ is intended to answer the questions of "inquiring breeders who, having more or less incorrect notions of what a geneticist is and does, yet seek assistance in the solution of their problems." The authors concluded that someone had "got to explain to the breeders of budgerigars what genetics is"; hence their book. They therefore forego such discussions as that of Steiner on the relation of budgerigar genetics to problems of evolution and settle down to the task of making the mysteries of genetics intelligible and usable to the interested but uninformed breeder of budgerigars. Unless, however, the fancier has some knowledge of genetics in advance, there may be some question as to how much of a usable grasp he will get of the subject from the presentation. Considerable space is devoted to an exposition of Mendelism on the basis of budgerigar inheritance, but the Drosophila symbolism of a + sign for "wild type" rather than a letter, while useful to the geneticist, is apt to be confusing to a beginner, especially when both chromosomes of a pair are sometimes represented in the diagrams and in other cases the symbols are put on the two sides of a single line. It may be questioned also whether the average fancier will be able to follow the different possible explanations of mosaics without some foundation knowledge of cytology and development. This is not because the material is not well presented, but is due to the inherent complications of the subject.

Whatever the practical breeder may get out of the book, and he will doubtless absorb a great deal even if he does not get it all, it will serve as an interesting and valuable summary to the geneticist. Should the latter desire

---

*_The Cult of the Budgerigar_. W. WATMOUGH. Illustrated with drawings and paintings by R. A. Vowles, and numerous sketches and photographs. Price: Six shillings net. London. viii + 296 pp. No date.

†_Vererbungsstudien am Wellensittisch, Melopsittacus undulatus_ (Shaw). Ein kasuistischer Beitrag zum Domestikations-problem. DR. HANS STEINER. Separatabdruck aus dem Archiv der Julius Klaus-Stiftung für Vererbungsforschung, Sozialanthropologie und Rassenhygiene. Zürich, 1932, Bd. VII, Heft 1/2 pp. 37-202, pls. I-X (colored).

‡_The Genetics of the Budgerigar_. F. A. E. CREW and ROWENA LAMY. Watmoughs Limited, Idle, Bradford, and London. x + 122 pp. Author's preface dated 1935.

more genetic detail he can go to Steiner, whereas if he is inclined to take up the breeding of budgerigars for himself he will find Watmough's book an excellent guide. Everyone accordingly appears to be taken care of for the present and the cult of the budgeriar should continue to increase and prosper.

L. J. COLE.

University of Wisconsin.

A DYNAMIC THEORY OF PERSONALITY

THIS volume consists of a collection of articles "written at different times and for quite different occasions." The selections discuss the psychology of the person and of the environment, touching upon the realms of child psychology, psychopathology, characterology, social psychology and pedagogy. There are eight chapters, an index of names, and an index of subjects.

Following an excellent review of the Aristotelian and Galilean modes of thought the author discusses the structure of the mind and its modes of dynamic adjustment to the environment. He points out that the current trend or ambition to demonstrate the scientific status of psychology by using statistics and as much mathematics as possible, and by pushing all calculations to the last possible decimal place, may be, in a way, laudable, but certain criteria combine into single groups cases that are psychologically very different or even antithetical. Such statistical investigations are consequently unable, as a rule, to give an explanation of the dynamics of the processes involved.

From a psychological standpoint the environment is sometimes understood to mean the momentary situation, and at other times the milieu or more or less permanent situation. Whatever the concept of environment may be, it is obvious that in every adjustment the individual is concerned with a complex aggregate of environmental events and predispositional factors. Inferences taken from the average or general run of reactions, and applied to concrete cases or situations, are invalid and have no practical utility, because a knowledge of the concrete total situation is necessary to evaluate the factors involved. "In psychology the forces of the environment and the laws of their operation on child behavior can be discovered only by proceeding from certain total situations that are simple but well defined in their concrete individuality."

As a dynamic system the individual is more or less unitary and self-contained. However, there are marked individual differences due to a diversity of structure in the total system, the degree of differentiation, the state of the system and the meaning of its content. There are differences in the degree of harmonious activity within the total structure, and in the ability to change, so the psychic material is said to have dynamic softness, elasticity, brittleness, fluidity, etc.

In Lewin's keen analysis of the dynamics of the relationship of the child to his environment at any given time, he has perhaps dealt too lightly with the multitude of factors which depend upon the internal structure and integration of the organism. Many of these factors depend entirely upon heredity, others upon the levels of development and maturation, still others represent modifications of previous experiences with the environment. All of these have been accumulated, integrated (thus changed), and carried forward into any present situation, truly a multiplicity of elements for consideration in addition to the stresses and factors that can be analyzed in the environmental situation. The author's emphasis on behavior as determined by stresses, drives, and shifting forces, rather than by what is known as

A Dynamic Theory of Personality. By KURT LEWIN, PH.D. (Translated by D. K. Adams and K. E. Zener.) Pp. ix and 286. New York and London, McGraw-Hill Book Co., Ind., 1935.

"absolutes," is a step forward in experimental psychology.

Lewin and others of the school of experimental psychology which he represents have developed ingenious techniques with which to study a wide variety of reactions in normal and abnormal individuals including motor activity, attitudes, attention, conflict, the emotions, fantasy, memory, hallucinations, and many others. The details of the techniques used are to be found in the original articles published. The general principles of arranging the experiments to apply to the type of activity under investigation deal with the structure of a definite situation, the interests involved and the attitude of the subject toward the experiments. From the results of these methods of study the author has formulated dynamic theories which point the way to a better understanding of child development, early conflicts and degrees of original capacities, which matters have a direct bearing on the problem of evaluating genetic and environmental factors in the development of personality. A chapter on reward and punishment and one on feebleminded-ness are particularly thought provoking.

N. D. C. Lewis
Neurological Institute of New York

✦

SOME QUESTIONS CONCERNING TWINS

To the Editor:

HOW many pairs of twins are represented in *Who's Who in America?* In the whole United States, twins occur about once in eighty births, and the ratio is not very different in other countries. So if twins live as long as other people they should constitute about one in forty, or 2½% of the population. And if they are just as likely to attain distinction as other people, there should be among the approximately 30,000 persons listed in *Who's Who* about 750 people who are twins, or about one to every third page (of the last few issues, which have three columns to the page).

But I have examined most or all of the pages of several volumes of *Who's Who,* digging out statistics of various kinds, without discovering a single *pair* of twins. Of course some of the men may have twin sisters who are married, and therefore listed on other pages, or some may have had twin brothers or sisters who died young, or never happened to distinguish themselves. But in a case of identical twins it would be strange if one attained distinction and the other did not. There are many cases of two or more persons in the same family in *Who's Who,* but just what proportion of all notables they constitute probably has not been calculated. An interesting study of relationships among scientists, by Dean R. Brimhall, ran through four numbers of the American Naturalist in 1922 and 1923.

I have found just one man in *Who's Who* whom I know to have had a twin brother, and I would not have known that if I had not gone to school with them in Massachusetts fifty years ago. But they were evidently not identical twins, for they differed considerably in disposition, and enough in appearance so that they would not easily be mistaken for each other.

Even if there should be a few undetected twins in *Who's Who,* they appear to be very much scarcer than their proportion at birth would indicate. This may be partly due to the fact that twins are said to be shorter-lived, on the average, than other people. Another possible explanation is this. The people listed in *Who's Who* represent only about one in 4,000 of the population of the United States, or about one in 500 of those of corresponding age (average about 56). So if only one child in 500 born attains such distinction, the chance of two notables being born at the same time should be very much smaller. (I will leave it to the mathematicians to figure out the probabilities.)

Another question is, how many grown-up negro twins are there? I do not recall ever seeing any, or reading about them in the newspapers, in a

residence of many years in the South. According to the official statistics, twin births occur more frequently among negroes than among whites. Taking 1933, the only year for which we have birth records covering the whole United States, 2.30% of the white children and 2.97% of the colored children born in that year were twins. Are negro twins so short-lived that few of them grow up? Infant mortality statistics do not seem to go into such details yet, but there might be some school enrollment records that would throw some light on the subject.

In copying names and dates from about 20,000 tombstones scattered from Massachusetts to Arizona, in recent years, I have come across comparatively few twins, perhaps 100 cases in all. (And no triplets that I can recall.) But that perhaps is not surprising, for if both twins grew up and married, or if they died several years apart, they might not be buried near together. A curious exception to the last is shown by a double tombstone in Thomasville, Georgia, for two twin girls, one of whom lived seven months and the other 23 years.

A most remarkable case of twins can be seen in the cemetery in Athens, Georgia—a boy and girl, born about two months apart, in different years (1882 and 1883), and both living several months. If I had noticed that in my student days (1894 to 1897), I might have found many people who remembered the circumstances; but I did not discover it until 1928, and was then unable to get any further information. But it might still be possible to find some discussion of the matter in newspaper files or medical journals of that period.

ROLAND M. HARPER.
University, Ala.

If inclusion in *Who's Who* is purely a matter of chance, and if twins have a frequency of 1/40 in the population, it follows that the chance of a *pair* of twins appearing in *Who's Who* would be 1/1600. On this basis we would expect only 18 *pairs* in a total of 30,000 entries. Since about a quarter of all twins are identical twins, it would follow by chance alone that about five pairs of identical twins would be expected in this volume, or one in 440 pages.

The editor knows of only one pair of identical twins in *Who's Who*—the Grosvenor twins, Gilbert and Edwin, whose astonishing similarity was described in the twin number of the JOURNAL (December, 1919). He has enough confidence in the ability of twins to hold up their end biologically as well as statistically, to offer a reward of ten dollars for the first list of ten same-sexed twin pairs, both of whom are included in *Who's Who,* and additional rewards of a dollar each for any additional same-sexed twins not on this list of ten. In the event that no list of ten is submitted, payment will be made at the rate of $1.00 per pair. Announcement of *Who's Who* twins "discovered" will be made in the JOURNAL.

The data given in *Who's Who* allow us to deduce that two individuals are twins only because of identity of parents and of birth dates. It gives no direct information as to this point. Therefore we can learn nothing from "the record" as to how many twins, with the other member not represented, are included in the volume, and the chance of locating female twins, where one member is married, is very slight.

Other identical twins who have attained scientific distinction are the Piccards, Auguste, the stratosphere ballooning pioneer, and Jean, the chemist and biologist. In *American Men of Science* are listed two of a set of triplets—Robert B. and Wallace R. Brode. It is of interest in connection with heredity of ability that the father of the Grosvenor twins is listed in *Who's Who,* and that the father of the Brode triplets also appears both in *Who's Who* and in *American Men of Science.* Even though twins should not be represented in *Who's Who* with the expected frequency we could not be too sure that this should be ascribed to biological inferiority. It should be born in mind that the method of selection of names

for inclusion in this volume may affect the result very materially. Certain groups are automatically included,—professors in colleges, government officials, civil and military, above a certain rank, etc., etc. Other activities requiring about the same intellectual proficiency do not lead as inevitably to a place in his volume. For that reason a closer parallel in twins' attainments may actually exist than would appear by the *Who's Who* test.—EDITOR.

✦

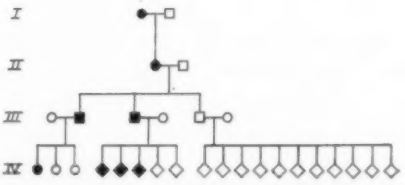
AN AMERICAN PEDIGREE EOR WOOLLY HAIR

EDGAR ANDERSON
Missouri Botanical Garden, Washington University, St. Louis, Missouri

ON a recent collecting trip in the South I met a young woman (IV-1 of the accompanying chart), the appearance of whose hair was similar to that illustrated in the papers by Mohr[1] and by Schokking.[2] Inquiry revealed that the condition had been known in her family for at least four generations and that it was apparently a simple Mendelian dominant, as in the cases reported by Mohr and Schokking. While the accompanying pedigree is short it illustrates several critical points: inheritance through both male and female, inheritance only through an affected individual, approximate equality of affected and non-affected in segregating sibships.

Since the family came from Old American stock it seems quite probable that other pedigrees will be found in this country. It may be useful in establishing kinship to record that II-1 was of a Southern family and that her maiden name was Metcalf.

To one accustomed to the woolly hair of negroes and many mulattoes, the peculiar hair exhibited by these pedigrees seems something quite different. It has an open, unmatted appearance and gives the impression of a peculiar kind of hair rather than of negro hair. As a matter of fact, I first saw the young woman who supplied the information from a passing automobile, but even at that distance recognized the condition as being similar, if not identical, with that illustrated in the JOURNAL OF HEREDITY.



FOUR GENERATIONS OF FRIZZLED HAIR

Figure 9

Pedigree chart of woolly-haired family. Solid black, woolly haired; outlines, normal hair. Circles represent females; squares, males; diamonds, sex unrecorded.

Literature Cited

1. MOHR, OTTO L. Woolly hair a dominant mutant character in man. *Jour. Hered. 23:* 345-353. 1932.

2. SCHOKKING, C. PH. Another woolly-hair mutation in man. *Jour. Hered. 25:* 337-340. 1934.

THE GENETICS OF DIABETES INSIPIDUS

And its Bearing on the Problem of Dominance in Man

S. G. Levit and L. N. Pessikova
Maxim Gorky Medico-Genetical Research Institute, Moscow

THE opinion is rather widespread (Lenz, Verschuer, Hogben, and others) that diabetes insipidus (which we will designate DI for convenience) is due to a dominant gene clear-cut in its expression. This view is based largely upon the well known case of Weil in which DI was transmitted for six consecutive generations. Similar cases in which the trait was transmitted for five generations have been reported by Chase, and Gänslen and Fritz. Hogben, on the basis of material gathered by Bulloch, calculated the number of sibs in the respective pedigrees and showed that the ratio of affected to healthy agreed with theoretical expectation, i.e., 1:1, but with a rather high observed number of affected.

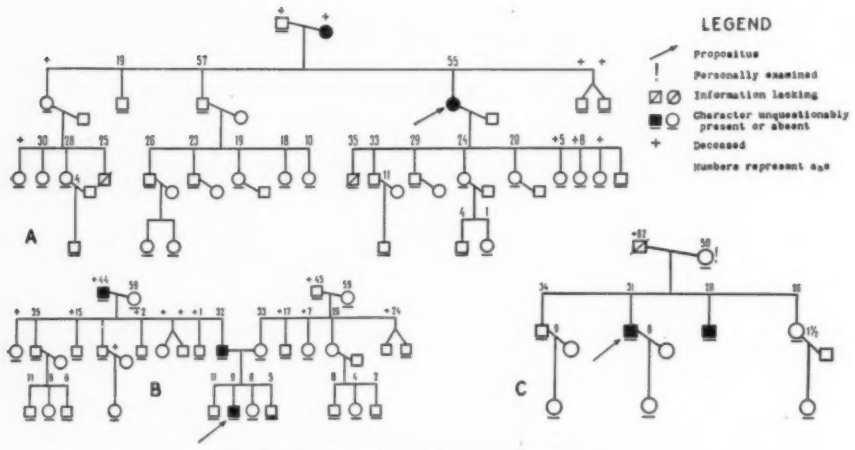
In the light of Levit's hypothesis that the majority of mutant genes in man are conditionally dominant, i.e., sometimes, but not always, expressed in the heterozygote, with the phenotype of the homozygote unknown, it seemed of interest to check the opinion of the above authors, particularly in view of the fact that the cases collected by Bulloch were not systematically gathered by their respective authors. It is known that such material has little significance for serious genetical analysis since the cases have usually been selected as "interesting," i.e., definitely familial. Furthermore, the conclusion that DI is a clear-cut dominant may be questioned, in view of the presence of skipped generations even in the cases gathered by Bulloch. Among the latter (13 familial cases) there are at least three skips. First there is the case of Weil, mentioned above, where there was one skip. In the cases of Gee there were three skips. In the case of Clay both parents of the three affected children were unaffected. There is a likelihood of skips in still other cases (Lacombe, McIlraith, and Ehrmann).

The chief symptoms of DI are polyuria and polydypsia. Affected persons drink large quantities of water and void similar amounts of urine of low specific gravity which does not increase with intake of large amounts of salt. The urine contains no sugar or albumin. No definite hypothesis has been established to explain the phenogenetics (pathogenesis) of this disease. Evidently there is a primary polyuria and a secondary polydypsia. In some cases, at least, the hypophysis is affected. The prognosis is usually favorable, the affected often living to old age.

The Present Material

Twenty cases of DI were investigated. Four were excluded from the calculations. Of the latter, three were not included because DI could be considered as a secondary condition (due to syphilis or to a tumor of the hypophysis). The other case was not included because the symptoms were atypical and the possibility could not be excluded that the polyuria and polydypsia were both secondary.

The pedigrees collected contained from 8 to 72 people. All propositi were thoroughly examined by the authors. X-rays of the sella turcica were taken. Chloride and sugar content of the blood were examined and an analysis of the urine was made. A water-free diet was given in every case. A similarly thorough investigation was also made of available relatives. The majority of the propositi were treated at the Medico-Genetical Institute. Case histories were carefully obtained for those examined by the authors elsewhere.



PEDIGREES OF DIABETES INSIPIDUS

Figure 10

Pedigrees of three families discussed in this article, one showing the irregular inheritance of the character (*C*). It is probable that a dominant gene showing poor penetrance is involved. By that is meant that in some instances even when genetic predisposition to the condition exists, it fails to be manifest. This may be due either to genetic modifying factors or to differences in internal or external environment.

Besides the propositi, 11 relatives were also personally examined. The preliminary data had been gathered from the propositi themselves, and our personal findings confirmed their reports. Thus it may be judged that the information concerning their relatives obtained from people affected with DI is, on the whole, satisfactory.

Clinically, the material can be separated into two parts, (1) 13 cases of typical DI, and (2) 3 cases where besides DI, dwarfism was also present.

Group 1

In 10 of the 13 cases there were no others affected besides the propositus. In the following we include an account of all affected individuals in the three familial case histories:

Case 3 (Figure 10*A*), ♀, 55 years old. From early age she drank very much and voided abundant urine. Thirst was marked especially during pregnancy. She used to get up frequently during the night to satisfy her thirst. At present she feels much better, although she still drinks about 10 glasses daily. Sella turcica normal. Wasserman negative. According to patient, her mother used to drink about a bucket of water daily and her thirst was also very strong during pregnancy.

Case 11 (Figure 10*B*), ♂ 31 years old. Since 1924 has suffered from pulmonary tuberculosis. In 1934 he became ill with exudative pleuritis and soon after began to have fearful thirst. He now drinks about 20 glasses of water daily and passes an excessive amount of urine. He often gets up during the night to quench his thirst and to pass urine. His condition has improved during the past months. The specific gravity of the urine is not higher than 1.011. Sella turcica normal and Wasserman negative. A brother of the patient had DI at the age of 22. The symptoms were similar to that of the propositus.

Case 15 (Figure 10*C*), ♂, nine and a half years old. According to patient, DI came on for no apparent cause. Drinks up to three liters a day and passes an abundant amount of urine. Specific gravity of urine 1.033, with no presence of albumin or sugar. Considerable anaemia. Sella turcica normal The grandfather first got DI at the age of 13 and used to drink as much as a bucket of water daily. He died at the age of 40 from typhus. The father of the propositus has suffered from DI since he was 16 years old. He, too, drinks as much as a bucket of water daily.

Group 2

There were no familial cases in this group, i.e., only one known case oc-

curred in each family. The statures of the patients in Group II were as follows:

Case 4, 7 years old, 102 cms.
Case 5, 13 years old, 117 cms.
Case 14, 24 years old, 117 cms.

Discussion

Our data are of interest for two reasons. First, they indicate very clearly the questionable validity (for purposes of genetic interpretation) of selected material gathered from the literature as compared with data that is systematically gathered without recourse to selected familial cases. From a study of Bulloch's data one gets the impression that every case is a familial one. In our material of 16 cases of DI only three were familial. Even if we exclude group 2 (i.e., DI accompanied by dwarfism), we nevertheless find, among 13 cases of DI, only 3 familial cases.

More important still is a second consideration. It seems apparent from our material that there is no reason for believing that DI is due to a clear-cut dominant gene. Just as in the majority of the other hereditary human diseases, it may be conceded here also, that the disease is due to a conditionally dominant gene (NB! there are no consanguineous marriages among the parents of our patients) showing poor penetrance.

One point is clear—it is hardly likely that all the sporadic cases are non-hereditary, or even genotypically different from the familial cases. This seems evident from the presence of skips in Bulloch's material. As a matter of fact, weshould, if we accept the hypothesis of a single dominant gene, also consider our Case 11 as containing a skip, since there were two affected sons born of normal parents.

Still other evidence could be introduced against a hypothesis that all sporadic cases are non-hereditary. If we accepted such a hypothesis we should expect that at least among the familial cases the ratio of affected children to healthy, issuing from matings between normal and affected parents, would be 1:1. As a matter of fact, in two of our familial cases* (Cases 3 and 15), the respective ratio among the children (if we include all children) was 3:21. The difference between the data obtained and theoretical expectancy is 9 ± 2.92, which makes such a hypothesis untenable. Moreover, even if we include only those sibs that have attained 15 years of age (although in our material there were a number of cases where the disease expressed itself in early childhood), we nevertheless obtain a ratio of two affected to ten unaffected, i.e., too few affected. There is hardly any doubt, therefore, that even in these last two familial cases the gene does not show complete penetrance. But if this is so, there is still less reason to suppose that the sporadic cases are exogenic ones. It is also unlikely that the sporadic cases are new mutations, as Hogben suggests, first, because mutations are rare, and secondly, even if we assume a high mutability for the locus in question as an explanation of these cases, still this hypothesis does not hold because of the fact that then the percentage of affected offspring from matings between normal and affected parents in our material would prove to be still less than when we consider the familial cases alone as we did above. Thus the ratio of affected to unaffected would be 3:37 if we include all the children and 2:21 if we include only those 15 years and older.

We are thus led to conclude that the development of diabetes insipidus is not due to a clear-cut dominant gene. The hypothesis of a conditionally dominant gene showing poor penetrance, seems, at present, plausible. However, the genetics of this disease needs further investigation. Further work along this line is now being carried on in our Institute.†

Literature Cited

1. Baur, Fischer, Lenz, Menschliche Erblichkeitslehre, III Aufl., 1927.

* Case 11 is not considered since it may be objected that the genetics of this case differs from that of the other two cases.

†The junior author has recently acquired new data for 70 cases of DI which substantiate the conclusions drawn above. This material is now being prepared for publication.

2. Bulloch, W., Diabetes insipidus. Treasury of hum. inheritance, vol. I, parts I-II, 1909.
3. Chase, L. A., cit. by Gates, Human Inheritance, 1929, London.
4. Clay, R. H., Three cases of diabetes insipidus in one family, *Lancet*, I, p. 1188, 1889.
5. Gänslen u. Fritz, Über Diabetes insipidus, *Klin. Wchnschr.*, H. I, 1924.
6. Gee, Samuel, cit. by Bulloch.
7. Hogben, Lancelot, Genetic principles in medicine and social science, New York, 1932.
8. ————————, Nature and Nurture. London, 1933.
9. Levit, S. G., Dominance in Man. *Compt. Rend. de L'Acad. des Sc. de L'USSR*, vol. II, N7, 1935.
10. Verschuer, Otmar, Erbpathologie, Dresden u. Leipzig, 1934.
11. Weil, A., Über die hereditäre Form des diabetes insipidus. *Deutsches Arch. f. klin. Med.*, B. 43, S. 181, 1908.

✦

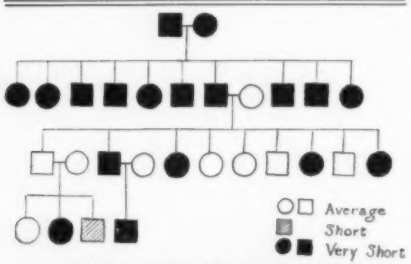
INHERITANCE OF SHORT, STUBBY HANDS

THE inheritance of short, stubby hands in this case has been traced through four generations. Actual measurements of the length and breadth of hands were made in all the members of the second and third generations but the hands of only three members of the first generation were measured. However, the hands of the other members of this generation are reported short by good authority. Therefore, *all* the sons and daughters of the progenitors of this line have short, stubby hands, as shown in the legend.

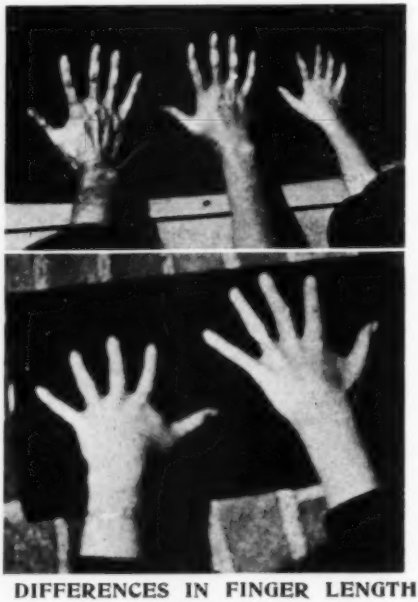
The mother of the second generation has hands of average length. Nearly fifty per cent of her offspring, however, have inherited the short hands of the father.

Two sons of the second generation are married and have children. In the family of the eldest son, one daughter, a child of five, has very short fingers and a wide, pudgy hand. (See Figure 1). In the second family, the child, a boy of eight, has very short fingers and a wide hand.

The short hands of the fourth son of the second generation, the hands of his oldest daughter and his second granddaughter are shown in Figure 12*A*. Figure 12*B* is a picture of the pudgy hands of his fourth daughter.



A SHORT-FINGER PEDIGREE
Figure 11



DIFFERENCES IN FINGER LENGTH
Figure 12

Above are shown three generations of a "short hand" family. The fourth son of the second generation, the first daughter of the third generation, and the second daughter of the fourth generation are shown. Below are shown the hands of the seventh member of the third generation, compared with that of a schoolgirl companion who had fingers of normal length.

Winifred Pendergast.
Mt. St. Scholastica College

TWO PEDIGREES SHOWING INHERITED PREDISPOSITION TO HERNIA

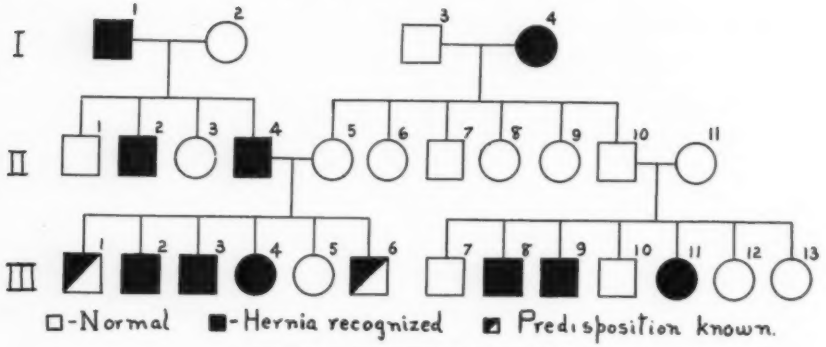
LUTHER S. WEST
Department of Biology and Eugenics
Battle Creek College

WHILE medical authorities have been prone to stress many factors other than heredity in explaining the occurrence of rupture, almost all modern literature on the subject makes recognition of some hereditary, or at least congenital, predisposition as a necessary basis for the development of the subsequent abnormality. Watson[14] in his "Etiology of Hernia" states that "the principal cause of hernia is undoubtedly the existence of a congenital sac. Other predisposing causes are: congenital weakness of the hernial ring, heredity, age, sex, pregnancy, obesity, trauma, and certain diseases." With respect to the importance of heredity this author states that 25 per cent of hernia patients give a history of hernia in their parents or grandparents, and quotes Macready[8] to show that male parents seem to transmit the predisposition more frequently to their sons than to their daughters, while ruptured mothers pass the weakness to their daughters more frequently than to their sons. This is explicable however, on the basis of the types of hernia transmitted, males showing inguinal hernia in the majority of cases, while in the female the femoral and abdominable types are more usual. Obviously, the structural conditions peculiar to each sex render the likelihood of inguinal hernia in the female, or femoral hernia in the male, comparatively slight. Assuming the inherited weakness in tissue structure to be somewhat specific as to location, children of opposite sex to the affected parent would naturally have good chances of escaping abnormal manifestations. Thus no special mode of inheritance need be involved or suggested to account for this tendency toward sex limitations in recorded cases. Watson refers specifically to a certain patient whose inguinal hernia was associated with partially descended testis. This man declared that both his father and paternal grandfather had the same condition. Quite certainly the sisters could not have manifested an anomaly so clearly dependent upon the abnormal location of the *male* sex gland. Such clearly marked sexual preference is not demonstrable however in the cases which we are about to consider.

In laboratory mammals and livestock considerable evidence on the heredity of hernia has been published. Moore and Schaible[10] have recently reported in this JOURNAL the occurrence of a high incidence of hernia in certain strains of rats. While the evidence of the hereditary nature of a predisposition to hernia is convincing, the exact mode of inheritance remains to be determined. In that paper references are given to other publications on the genetics of hernia in mammals.

Pedigrees Studied

Two pedigrees are presented, the first representing a family given largely to laborious pursuits, both out of doors and in factories. The second family history includes for the most part individuals of sedentary occupation, many of them being engaged in professional life. The frequency of hernia in the latter group is quite as great as in the first, a fact which tends to indicate that inherited conditions frequently manifest themselves regardless of occupation or environment, and that with respect to hernia at least, we need not subject the individual to severe and continual muscular strain in order that his constitutional proclivities may be disclosed. We



HERNIA IN THE *M* FAMILY

Figure 13

In this family, many of whose members engaged in active outdoor occupations, hernia has appeared for three generations. The larger number of individuals having or predisposed to hernia in generation III in the fraternity at the left is possibly due to an inherited tendency from both father and mother.

shall consider first the pedigree of the *M* family.

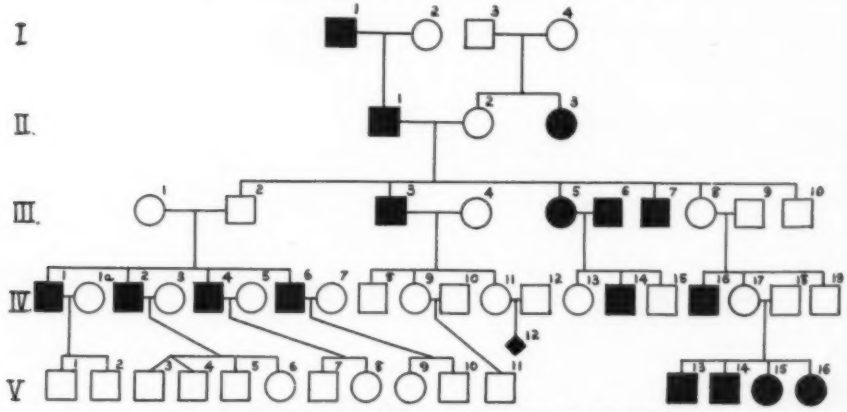
A Family in Which Physical Occupations Prevail

Individual III_3, who has been for some time in the author's employ and with whose condition he is most conversant, has the following record: He was born with right femoral hernia well developed, and wore a truss from babyhood until about the age of ten years. At this time, due to a boyhood prank for which a cousin was responsible, the truss was lost in a brook and never recovered. He has never since worn any support, though the condition gives trouble from time to time. C. M. is now 21 years of age and capable of almost any form of physical labor. He was twice examined in connection with the C.C.C., once at the county seat and again at camp, and in both cases was passed as normal, though the examining physician remarked that his abdominal wall was of a type likely to suffer rupture. The abnormality manifests itself when attempt is made to lift a heavy object directly off the ground. Pain and general weakness are at once noticeable. A small bulge occurs which C. M. is able to readily adjust himself. After about a half-hour's rest he is again fit for labor and apparently suffers no ill effects from the experience.

The affected brothers and sisters of C. M. each have peculiar characteristics not shared by the others. The eldest brother, Robert (indicated in the pedigree chart as III_1), is now twenty-eight years of age. Robert is not actually ruptured but has a relaxed condition in both inguinal rings and the tissue adjacent, and has been repeatedly warned when examined in connection with factory employment. He is an expert welder.

The second brother, Maynard (indicated in the pedigree chart as III_2), is a young man of unusual strength. He was born with a right femoral hernia which bulged as large as a "hen's egg," and which failed to reduce naturally. The condition more than once threatened his life as an infant. A truss was worn until three years of age, at which time the condition was apparently adjusted and the truss abandoned. There has been no surgery and no return of the hernia. As stated previously, M. M. is unusually active and strong. He is 25 years of age and engages largely in factory labor of a heavy type.

The fourth child, Juanita (III_4), is a rather interesting case. So far as known, she displayed no hernia of any kind at



INHERITANCE OF HERNIA IN THE *G* FAMILY

Figure 14

In this family, with many members in professional or sedentary occupations, the incidence of hernia is about the same as in the *M* family. The pedigrees of these two families suggests that in man as in several other mammals an hereditary predisposition to this condition exists.

birth. At three years of age she fell down the cellar stairs and induced a right femoral hernia which was supported by a home-made truss. A year later while jumping from a platform to catch a swinging rope, she acquired a *left* femoral hernia very similar to the first. She wore a double truss for the next five years, at which time the rupture on the left was satisfactorily reduced. The right femoral hernia still persists, however, and is supported by a truss. J. M. (III_4) is a young woman of athletic and wiry type. She is now 18 years of age and has recently married.

The two younger children, Betty (III_5) and Kenneth (III_6), aged 14 and 9 respectively, have as yet no positive record of abnormality. The author, however, feeling that it might be possible to detect a predisposing weakness, arranged for an examination by a disinterested physician (Dr. B. M. Overholt). His signed statement is given herewith:

Examination of Kenneth M.

There appeared to be unusual weakness of the tissues of the inguinal region on both sides. Coughing gave a marked pulsation. On examination the external inguinal rings were found to be much larger than is normal for a boy of his age, and easily admitted the index finger.

Examination of Betty M.

Examination of the inguinal and femoral areas revealed no abnormalities.

On the basis of these findings we see that Kenneth is obviously predisposed to hernia. He will in fact be exceedingly fortunate if he goes through life without it.

The mother of this family of six is a normal, active, vigorous woman without any history of rupture. Her mother, however (I_4), has a positive history which is rather unusual. Etta S. was very muscular as a young woman. She was married early and had her first child at the age of 19. As a result of childbirth a serious femoral hernia appeared on one side. A single truss effectually supported the weakness until some 12 years later, when a similar condition was acquired on the opposite side. It is said that the second hernia resulted from the effort of "lifting a cow out of a spring." (In all probability the procedure consisted of pulling on a rope tied fast to the mired animal. In any event, the record has it that the cow was successfully extricated from her precarious position.) E. S. has worn a double truss since that time, and is unable to do without it even for short periods. She is 65 years of age,

and otherwise vigorous.

II_4, the father of the six children is a tall, spare, active man of 48. His principal occupations have been farming and butchering, together with some factory work which proved responsible for the development of his abnormality. At the age of 35, while attempting to prevent the slipping of a heavy piece of metal which he was carrying against the abdomen, B. M. suffered an abdominal rupture just above the navel. A further attempt at similar labor rendered the condition double in character and completely incapacitated the patient. A surgical operation performed very soon after this was entirely successful in restoring the normal condition. There has been no recurrence and there are no after-effects beyond an occasional stiffness following fatigue.

B. M.'s brother Howard (II_2) was born with a right femoral hernia. During childhood he wore a truss, which seemed to effectually reduce the condition. He is now 36 years of age and has had no recurrence.

Levi M., the father of these men (I_1), now deceased, had no evidence of hernia in youth, but developed a single femoral hernia in middle life in connection with unusually strenuous labor. He wore a truss until his death at the age of 60 years.

There are three cousins on the mother's side of the family, shown in the pedigree as III_8, III_9, and III_{11}, who are affected. Robert S. (III_8) was believed to have developed his right femoral hernia as a result of hard crying while less than one year of age. He wore a truss until three years of age, after which the truss was abandoned. He is now 20 years of age, a cripple, hence inactive and therefore not liable to experience further difficulty. The brother, Dale (III_9), has a similar early history, having ruptured himself shortly after birth, if the condition was not truly congenital. His hernia was also right femoral. He wore a truss for two or three years, after which he appeared normal. He is now 18 years of age, strong and vigorous, and has had no recurrence. The sister, Beulah (III_{11}), is known to have a right femoral hernia, but detailed information as to her personal history is not at present available.

Pedigree of the "G" Family

The second family studied, including as it does a number of individuals of *professional* and *sedentary* pursuits, is quite as interesting as the first, especially so since the types of hernia manifested are altogether different. In the *M* family, femoral and abdominal hernias were the rule and there was no record of umbilical hernia in any case. In the *G* family, however, inguinal hernia is predominant, and umbilical hernia frequent, while femoral hernia is unknown. A single case of abdominal rupture (III_3) apparently relates to unsuccessful surgery and is probably not to be attributed to constitutional factors.

Caleb G. (I_1) was a skilled iron worker and blacksmith. He did much heavy lifting and acquired a single inguinal hernia some time in early or middle life. He wore a truss all his life, and died at 78 years of senility hastened by bladder trouble.

Caleb's son, William (II_1), was a printer and also a minister. As a young man he suffered with a left inguinal hernia and wore a truss for many years, which, however, he abandoned in later life. He died of paralysis agitans at the age of 68.

There is no other positive case among the older generations except that of William's wife's sister, who has already been referred to as probably exceptional. She developed a combined inguinal-abdominal condition as a result of a laparotomy performed at the age of 50. She lived to the age of 74.

Of the three generations known to the author, the first two show a generous sprinkling of the family weakness. The last generation is made up of children who are for the most part very young; and since few of the ancestors (except for umbilical conditions) acquired their hernias before late adolescence, we need not be surprised that in

generation V, only umbilical hernias have, as yet, been recorded ($V_{13, 14, 15, 16}$).

Frank G (III_3), who spent most of his life as a printer and engineer, developed a single inguinal hernia (on which side is not recorded) while still a young man. He wore a truss all his life; died of a heart attack at 64.

Frank's sister, Linda (III_5), a physician, developed a left inguinal hernia at the age of 12, from jumping. She wore a truss for six years, and then, desiring to be admitted to a nurses' training school, submitted to surgical repair. This was thoroughly successful and she is now vigorous and active at the age of 62.

While not of the same blood line, it is interesting to note that Linda's husband, P. R., is also ruptured, a condition which may have a bearing on the appearance of hernia in one of their children. P. R. (III_6), also a physician, lifted an oxygen tank at the age of 45 and developed an inguinal hernia on either side. He had surgical repair for the right, but had such difficulty in surviving the anaesthetic that no attempt was made to secure the condition of the left. He is now over 64 and still needs surgical relief for the left hernia, but his idiosyncrasy to anaesthetics renders this undesirable.

Linda's brother, Harry (III_7), who died at seven years, had a double inguinal hernia which was either congenital or acquired immediately after birth. He wore a truss until his death.

In generation IV we find an interesting concentration of hernia among the children of Fred G. (III_2), who has never himself been ruptured though his brothers and sisters were variously affected, as has already been pointed out. All four of Fred's sons were born with umbilical hernias.

The oldest son, Harry (IV_1), has never developed any other type of hernia, but each of the remaining three have.

The second son, Edwin (IV_2), has already had surgical repair for one inguinal hernia, which did not prove successful. He has since developed a second inguinal hernia on the opposite side and must now have surgical repair for both. He is between 35 and 40 years of age.

The third son, Walter (IV_4), has had inguinal hernia on both sides for some time and is now about to be operated on.

The fourth son, William (IV_6), developed his first inguinal hernia at 19, and submitted to surgical repair soon after. His second, on the other side, developed at 32. This also received successful surgical attention.

Two cousins of these men have also been ruptured. Herman R. (IV_{14}), who had umbilical hernia at birth, first noticed his inguinal hernias at about the age of 21. The right hernia was complete, the left only partial. Surgery accomplished the successful correction of both. He is now 30 years of age. The second cousin, Elmer (IV_{16}), displayed an umbilical hernia at birth and a double inguinal became apparent immediately after. He was a premature child of only seven months' gestation. A special truss of "yarn" was devised, by means of which these were effectually reduced and supposedly cured. One broke open, however, at the age of 20 when the subject was a student in medical school. The second likewise reasserted its presence when the subject was 27 years of age and engaged in medical missionary work in China. Each received surgical attention shortly after diagnosis.

The four children of this man's only sister were all born with umbilical hernia. They represent, as yet, the only manifestation of inherited abnormality in Generation V.

Discussion and Interpretation

Both femoral and inguinal, as well as abdominal hernia, are essentially similar in that all are merely protrusions of a loop or knuckle of an organ from its normal position, either into the inguinal canal or through the abdominal wall. The rather widespread occurrence of one or another of these conditions may well be a matter of concern, especially as it affects the efficiency of those en-

gaged in physical pursuits or inclined to participation in athletic sports. We find in Bauer, Fischer, and Lenz[2] the rather striking statement that one in every 20 or 30 men rupture themselves at some time in life. The figure is far lower for women, being given as one in 150. Were it not for the structural differences between the sexes, such figurs might easily indicate a sex-linked mode of inheritance, but apparently no authors have taken seriously this interpretation. The above mentioned authors assert that *dominant* hereditary factors control the predisposition to this complaint, and heredity is stated to be of at least as great importance as the aggregate of all exciting causes pertaining to the onset of the trouble.

Castle[4] affirms the hereditary nature of predisposition to hernia but declines to suggest by what mendelian principle the transmission takes place. Guyer's statement[6] inclines toward an interpretation based on the assumption of recessive genetic factors. The present example, though not inclusive of a sufficient number of individual cases to be of statistical value, nevertheless strongly suggests the operation of a mendelian dominant in at least certain branches. In the case of the "M" family the abnormality is clearly present in something over half the individuals making up the pedigree on the father's side, and in the same branch there is no skipping of generations. On the other hand the mother's side of the pedigree shows a skipping of the intermediate generation at two separate points. Turning to the rather more prolific "G" family, we find the apparent normality of Fred G (III_2), and of his sister Mabel (III_8), interrupting an otherwise continuous transmission of hernia. Of course if all the apparently normal persons in these pedigrees could be subjected to a careful examination, as were the two children (III_5 and III_6), many would, in all probability, betray an inherent weakness in the rupture area, even though they may be so fortunate as to go through life without precipitating the actual condition itself.

Therefore, even though we are scarcely prepared to state with certainty the exact mechanism by which heredity operates in this condition, the recognition of an inherited factor in hernia is undoubtedly substantiated by the study of such pedigrees as these.

The prevailing attitude of medical men as well as anthropologists ten to twenty years ago seemed to be marked by an effort to exclude all hereditary influence, if possible, in the interpretation of human abnormality or disease. The prevalence of umbilical hernia in certain Polynesian people was attributed exclusively to obstetrical technique. Poor physical development and laborious occupations were likewise held responsible for racial tendencies to hernia in other groups. The prevalence of intestinal hernia, especially of the caecal type among the people of India, was sincerely and seriously interpreted by Thurston[12] and Connor[5] as entirely due to the effects of a bulky vegetarian diet. Such food habits were believed by them to cause undue enlargement of the intestine, resulting in much greater likelihood of displacement than would be possible in the case of meat-eating races. This is quite in contrast with the present period in which the salutory effect of a considerable amount of vegetable bulk in the dietary is generally acknowledged, and the health and tone of the intestinal wall and its supporting mesentery, is believed to be strongly favored by a bulky content with its natural stimulation of peristaltic action.

We should, in all our interpretations, remember that even though a condition be described as *very common* in a given population, it is scientifically unsound to even suggest that the condition is the direct result of the prevailing habits of the people unless a majoriy of the individuals practicing such habits actually display the trait. And even should such a convincing state of things be demonstrated, there must ever remain the problem of accounting for those few or many, as the case may be, who, though following all the prescribed procedure for the attainment of the specific abnormality, fail to achieve that

end. The constitutional peculiarities of the individual offer the only possible explanation which is in harmony with all the facts. Lastly we should bear in mind that, although these hereditary differences exist, potentially, from the beginning, they may or may not ever become manifest in individual lives. No man ever is subjected to environmental experience so diverse that the *entire* genetic equipment which he possesses can come into full display.

Literature Cited

1. Barker, L. F., and Pincoffs, M. C. Visceroptosis or Splanchnoptosis, umbilical and epigastric hernia. *In* Tice, "Practice of Medicine," which see .
2. Bauer, Fischer and Lenz. Human Heredity. Translated by Eden and Cedar Paul. The Macmillan Co., New York, 1931.
3. Capelle. Dauerresultate nach operationen der Hernia epigastrica. *Beitr. z. klin. Chir. Tübing.* 63:264-285, 1909.
4. Castle, W. E. Genetics and Eugenics. Harvard University Press, Cambridge, Mass., 1930.
5. Connor, E. P. Notes on eighty-five consecutive cases of strangulated hernia. *Indian Med. Gaz.*, 45:447-452, Calcutta, 1910.
6. Guyer, M. F. Being Well-Born. The Bobbs-Merrill Company, Indianapolis, Ind., 1927.
7. Lindenstein. Zur Lehre von der Hernia epigastrica. *Beitr. z. klin. Chir. Tübing.* 58:293-314, 1908.
8. Macready, J. F. C. H. A Treatise on Ruptures, London, Griffin and Company, 1893.
9. Mohr, O. L. Heredity and Disease. W. Norton Co., Inc., New York, 1934.
10. Moore, L. A., and P. J. Schaible. Inheritance of Umbilical Hernia in Rats. *Jour. Hered.*, 27:273-280, 1936.
11. Terrier, F. Surgical intervention in non-strangulated epigastric and adumbilical hernia. *Ann. Surg.*, V:374-376, St. Louis, 1887.
12. Thurston, E. O. Gangrenous strangulated inguinal hernia. *Indian Med. Gaz.* 39:16, Calcutta, 1904.
13. Tice, F. Practice of Medicine. W. F. Prior Co., Inc., Hagerstown, Md., 1921.
14. Watson, L. F. Hernia—its anatomy, etiology, symptoms, diagnosis, differential diagnosis, prognosis and operative treatment. C. V. Mosby Co., St. Louis, 1924.

✦

Books Received

BOOKS are acknowledged in this column as received, and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. As far as space permits, books that contain material of special interest to the readers of the JOURNAL *will be reviewed in later numbers.*

TRAINING OF THE NEUROLOGIST AND THE PSYCHIATRIST, Reprinted from the Archives of Neurology and Psychiatry, Volumes 29 and 30, 1933, and Volumes 31 and 32, 1934. Copyrighted by American Medical Association. Pp. 102.

THE RELATION BETWEEN MORALITY AND INTELLECT, A Compendium of Evidence Contributed by Psychology, Criminology, and Sociology, *by* Clara Frances Chassell, Ph.D., formerly School Psychologist in the Horace Mann School, and Instructor in Experimental Education, Teachers College, Columbia University. Teachers College, Columbia University, Contributions to Education, No. 607. Pp. 556. Price, $4.50. Bureau of Publications, Teachers College, Columbia University, New York, 1935.

Wood's correlation between brains and social virtue remains not much changed in the light of compendial reconsideration.

THIRTY-THIRD ANNUAL REPORT (1934-1935) OF THE IMPERIAL CANCER RESEARCH FOUNDATION, Under the direction of the Royal College of Physicians of London and the Royal College of Surgeons of England. Pp. 35. London, 1935.

A HUNDRED YEARS OF ANTHROPOLOGY, *by* T. K. Penniman, M.A. (Oxon), Secretary to the Committee for Anthropology in the University of Oxford. Pp. 400. Price, $4.50. New York: The Macmillan Co., 1936.

A comprehensive survey of a Century of Anthropological Progress, during which time man has come to see himself as a citizen of this earth, with a proud history extending back to the time when he needed gillslits, rather than as a rather smug Star Boarder (who appeared on the scene complete even to the extent of such curious impedamenta

as the before mentioned gillslits and a vermiform appendix) on a spring morning in 4004 B. C.

EVOLUTION, *by* A. FRANKLIN SHULL, Professor of Zoology, University of Michigan. Pp. 312. Price, $3.00. New York and London: McGraw-Hill Book Co., 1936.

THE EGGS OF MAMMALS, *by* GREGORY PINCUS, Assistant Professor of General Phyiology, Harvard University. Pp. 160. Price, $3.75. New York: The Macmillan Co. 1936.

The Greeks couldn't decide whether the fowl or the egg came first. Biologists face an even more perplexing problem—to try to understand the miracle whereby is concealed in so tiny a speck of protoplasm such tremendous growth vigor, controlled by such profound developmental wisdom. Dr. Pincus ably Baedickers us about the frontiers of 1936 knowledge regarding this fascinating problem.

CIRCUS BOY, *by* HARRIET F. BLUM. Pp. 194. Price, $1.75. New York: The Macmillan Co. 1936.

A children's story-book recounting the adventures of a circus child. The only genetic or eugenic significance would seem to be a pointing of the moral that children may learn much by actually taking part in the adult world.

A LABORATORY MANUAL IN GENERAL BIOLOGY, *by* JAMES W. MAVOR, Professor of Biology and Chairman of the Field of Biology, Union College, and Leonard B. Clark, Assistant Professor of Biology, Union College. Pp. 201. Price, $1.75. New York: The Macmillan Co. 1936.

HOW WE CAME BY OUR BODIES, *by* CHARLES B. DAVENPORT. Pp. 401. Price, $3.75. New York: Henry Holt and Co. 1936.

HEALTH GUIDES AND GUARDS, *by* FRANCIS P. WALL *and* LOUISE D. ZEIDBERG. Pp. 208. Price, $1.35. New York: Prentice-Hall, Inc. 1936.

GONE WITH THE WIND, *by* MARGARET MITCHELL. Pp. 1037. Price, $3.00. New York: The Macmillan Co. 1936.

A most interesting and moving study of the diverse reactions of personalities to overwhelming disaster. The picture of "Security" in the South of 1860, serving as a backdrop for a tragic decade of Civil War and "Reconstruction" may have a 1936 moral for us who hope to attain security in the face of a suicidal differential birthrate in a creaking economic system, and to the tune of rattling sabres among the nations of the world.

BULLETIN OF THE NEUROLOGICAL INSTITUTE OF NEW YORK, Volume V. Elsberg Anniversary Number. Pp. 544. Neurological Institute, New York. 1936.

METHODS OF STATISTICAL ANALYSIS, *by* C. H. GOULDEN, Senior Cereal Specialist, Dominion Rust Research Laboratory, and Honorary Lecturer in Mathematics, University of Manitoba. Pp. 165. Price, $3.00. Minneapolis, Minn.: Burgess Publishing Co. 1936.

EL SOLANUM TUBEROSUM, a traves del desenvolvimiento de las actividades humanas (Contribution al estudio de la Planta Nacional), *by* CESAR VARGAS C. Pp. 73. Lima: Imprenta del Museo Nacional. 1936.

DIE DEFEKTSCHIZOPHRENEN KRANKHEITSBILDER, Ihre Einteilung in Zwei Klinisch und Erbbiologisch Verschiedene Gruppen und in unterformen vom Charakter der Systemkrankheitem, von K. Leonhard. Pp. 134. Price, M 8.70. Leipzig: George Thieme. 1936.

HEADS AND TALES, *by* MALVINA HOFFMAN. Pp. 416. Price, $5.00. New York: Charles Scribner's Sons. 1936.

A distinguished sculptress compiles a "Hall of Man," and in doing the job sees much of the world, and has no dearth of adventures in doing so. The text and the illustrations are alike fascinating.

KONSTITUTION UND AFFEKT, von Gerhart D. Mall, mit einem Geleitwort von Prof. Dr. Oswald Kroh. Pp. 104. Price, RM 8.60. Leipzig: Verlag von Ambrosius Barth. 1936.

WHY QUIT OUR OWN? *by* GEORGE N. PEEK with SAMUEL CROWTHER. Pp. 353. Price, 50c. New York: D. Van Nostrand Co., Inc. 1936.

ANNUAL REPORT OF THE BOARD OF REGENTS OF THE SMITHSONIAN INSTITUTION, showing the Operations, Expenditures, and Conditions of the Institution for the Year ending June 30, 1935. Pp. 580. Washington: Smithsonian Institution. 1936.

Containing, of particular genetic interest, Dr. T. H. Morgan's address, "The Relation of Genetics to Physiology and Medicine," delivered on the occasion of the Nobel Presentation to Dr. Morgan, at Stockholm, June 4, 1934.

"HEREDITY" LANTERN SLIDES

MAN

16-397. Gregor Mendel's Handwriting — First Page of "Studies of Plant Hybrids".
17-435. Merton House—Home of William Bateson.
21-1-Cover. The "Fuchsia-Picture" of Mendel.
21-No. 2-Cover. Francis Galton.
26-158. Ishmael Family—Six Generations of a Degenerate Family.
26-159. Roosevelt Family—Pedigree Chart of a Superior Family.
26-292. Dr. and Mrs. Hugo de Vries.
15-330. Fused Finger Joints.
17-393. Short Legs—Inherited Achondroplasia.
20-75. Extra Fingers—Recessive Polydactylism.
20-74. Chart Showing Inheritance of Polydactylism.
20-352. A Folding Man — Hereditary Absence of Clavicles.
22-104. Crooked Arms and Legs—Hereditary Bone Fragility.
23-307. Five Generations of Glaucoma.
23-425. Inherited Doublejointedness in the Thumb.
24-269. Chart Illustrating Sex-Determination.
22-47. Palm Prints of Fraternal Twins.
22-40. Corresponding Fingerprints of 3 Pairs of Identical Twins.
23-369. Identical Twins—Separated 13 Years.
23-2. Identical Twins Reared Apart—Mary and Mabel when Grown.
23-5. Identical Twins Reared Apart—Mary and Mabel as Babies.
23-6. Identical Twins (Mary and Mabel) at Two Years—Reared Apart.
23-8. Palm Prints of an Identical Twin (Mary).
23-9. Palm Prints of an Identical Twin (Mabel).
24-443. Mirror-Imaging in Eye Dominance in Identical Twins.
24-444. Table Classifying Handedness in Twins.
25-140. Table of Physical Characteristics of Identical Twins Reared Apart.
25-141. Identical Twins (Harold and Holden) Reared Apart.
25-293. Retina of Normal and Albino Eyes.
25-332. Abnormal Fingers and Toes — Syndactyly, Camptodactyly, and "Hammer Toe".
25-333. Short Finger (Brachymetapody) due to short metacarpal bone.
25-391. Hereditary Absence of Incisors.
25-446. Inherited Chest Depression—Koilosternia.
25-483. Sex-Linked Toothlessness.
25-338. Fifty-fifty Segregation for Woolly Hair Mutation.
26-143. Extra Toes—Inherited Polydactylism.
10-389. Twin Acrobats—Identical Twins.
10-409. Typical Identical Twins.
20-50. Identical Twins Reared Apart.
20-52. Palm Patterns of Identical Twins.
20-155. City Boy and Country Boy—Identical Twins Reared Apart.
20-160. Enlarged Prints of Left Ring Finger of Identical Twins (C & O).
22-202. The Hilton Siamese Twins.

ANIMAL

16-154. Pelvic Bones of Normal and Rumpless Fowls.
16-352. Leg Bones of "Reptilian Fowl" and of Normal.
19-413. A Fertile Mare Mule.
19-414. "Old Beck" (Mule) and Her Horse-Like Colt.
19-415. Mule-Like Colt of Fertile Mare Mule.
20-112. Recessive Leg Feathering in Fowl.
20-113. "Heel Tufts" in Fowl.
21-323. Heterogeneous Chromosome Structure — Irregular Cell Division in Drosophila Melanogaster.
22-213. Conjoined Twins in Cattle.
22-296. Backcrosses between Hybrid (Zebu × Yak) Female × Zebu Male.
22-298. Zebu Bull Used in Yak Crosses.
22-299. Yak Cow Used in Zebu Crosses.
22-305. A Zebu-Yak Hybrid—4 Years Old.
22-305. Zebu-Yak Hybrid Cow—4 Years Old.
21-449. A "Flightless" Rooster.
24-183. Parallel Color Mutations (Himalayan and Siamese) in Rabbit and Cat.
25-208. Five Generations of Inbreeding—Shorthorn Cattle (chart).
25-266. X-Ray Photos of Normal and "Flightless" Feathers in Fowl.
25-435. Production of Agglutinins in Rabbits (chart).
25-432. Agglutination of Incompatible Bloods.
25-464. "Portraits" of Salivary Gland Chromosome.
25-466. Giant Chromosomes Compared with "Normal".
25-469. Synapsis of Normal and Inverted Chromosomes.
25-470. Synapsis of Deleted and Normal Chromosomes.
25-472. Cyto-Genetic Maps of Salivary Chromosomes. (Insert.)
26-62. Salivary Chromosomes and Gonial Chromosomes Compared.
26-62. Reference Map of the Salivary Chromosomes of Drosophila. (Insert.)
26-182. Structure of Salivary Gland Chromosome.
26-301. Effects of Sex Hormone Injection on Feather Growth.

PLANT

15-204. Capsules of Globe Daturas.
17-372. Awn of Manchuria (Rough) Barley.
17-373. Awn of Lion (Smooth) Barley.
18-128. Adaptive Cupped Tendrils of a Plant.
18-129. Adaptive "Vacuum Cup" Toes of a Toad.
18-382. Leaves of Live Oak × Post Oak Hybrids.
18-383. Leaves of Overcup × Live Oak Hybrids.
19-419. "Fatback" and "Razorback" Honey Locust Pods.
20-24. Haploid and Dwarf Diploid Tomato Plants.
20-25. Haploid and Normal Tomato Plants.
20-26. Haploid and Diploid Tomatoes.
20-206. Flax Hybrid Produced by Artificial Nutrition.
22-368, 370. Plant Patents No. 2 and No. 3.
24-131. Radium-Induced "Ridged" Tomato.
24-133. Chimeral Radium-Treated Tomato Leaves.
24-134. Radium-Induced Virescent-White Tomato Seedlings.
24-368. Gene Mutation Chimera in Delphinium.
26-137. Ears of Maize, Relatives, and Hybrids.
25-448. Fasciated Sugar Beet.

Above are listed ninety Lantern Slides from JOURNAL OF HEREDITY illustrations. These are only a sample of over five thousand charts and photographs which have been published in the twenty-six volumes of the JOURNAL. Order from this list by Number. Slides may be ordered for any illustration appearing in the JOURNAL by giving Volume—Page—and Figure Number. Slides on this list will be supplied within a week of receipt of order. Price 75c each —$8.00 per dozen. Slides are numbered according to volume and page on which they appear. Thus 25-372 is from Volume 25, page 372 of the JOURNAL, 24-300 is from Volume 24, page 300, etc. Suggestions for illustrative material from the JOURNAL suitable for specific lectures will gladly be furnished on request. Address orders and inquiries to:

AMERICAN GENETIC ASSOCIATION

Victor Building **Washington, D. C.**

ANNALS OF EUGENICS

THE ONLY JOURNAL PRINCIPALLY DEVOTED TO STATISTICO-GENETIC STUDIES OF INHERITANCE

Edited by R. A. Fisher

(FOUNDED BY KARL PEARSON)

Vol. VII, Part 2, will contain articles by

L. McMullen
M. N. Karn
R. A. Fisher
F. Yates
A. S. Wiener et al.
S. Barbacki

SUBSCRIPTION PRICE 50/- PER VOL.

Address Your Order To

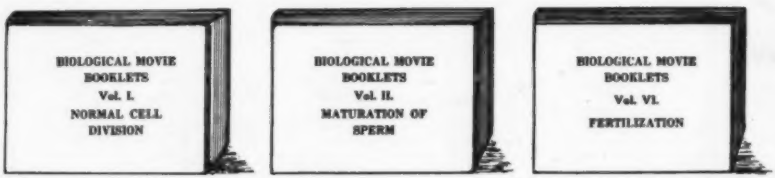
GALTON LABORATORY,

University College, Gower Street, London, W. C. 1.

THE BIOLOGICAL MOVIE BOOKLETS

DRAWN BY

DR. CLYDE E. KEELER



The set of three booklets—$1.50

(Separately Vol. I—50c, Vol. II—70c, and Vol. VI—60c.)

Can be obtained only from

AMERICAN GENETIC ASSOCIATION

VICTOR BUILDING WASHINGTON, D. C.